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DYSTROPHY OF THE CORNEAL ENDOTHELIUM

ITS RECOGNITION AND CLINICAL SIGNIFICANCE *

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The title of my paper may not appeal to many who are practically minded and who are not users of the slit-lamp in daily routine practice. The subject, however, is not an ultrascientific one but pertains to a practical clinical entity, a condition that is more widespread than even the cases herein reported would indicate, the cases having been gathered in routine study during the last eighteen months. Furthermore, the condition can be recognized by any one who uses the ophthalmoscope discriminatingly, although confirmation by the slit-lamp is convincing and illuminating. It has important clinical aspects and, in my judgment, has a bearing on operative results in patients affected with it when subjected to cataract extraction, and possibly to other intra-ocular surgical procedures.

Little has appeared in the literature on endothelial dystrophy or on endothelial pathologic processes in general since the rather exhaustive studies made by Basil Graves [1] in 1924. Brief references have been made by Vogt, Koby and others, but they have added little to Graves' exhaustive studies, and these were largely from the standpoint of the slit-lamp. Unless careful search is made for the condition clinically, one would gather that the phenomenon is of rather rare occurrence and not a large contributing factor in pathologic processes of the eye. It is surprising, however, how a careful search will disclose a considerable number of cases in the course of an average practice. Like other conditions, the slit-lamp has made possible the clinical recognition of this interesting phenomenon which was only partly understood before the advent of the corneal microscope.

Among the changes noted in a careful examination of the endothelium by means of the slit-lamp, there are three outstanding conditions which, although totally dissimilar, must be differentiated in making a diagnosis of endothelial dystrophy. These conditions are: first, the endothelial changes that are observed after an iritis or iridocyclitis; second, traumatic endothelial disturbance, and third, true dystrophy of

* Submitted for publication, June 15, 1931.

* Read before the Pacific Coast Oto-Ophthalmological Society, May 29, 1931.

1. Graves, Basil: Brit. J. Ophth. 8:502, 1924.

the corneal endothelium. Each of these conditions must be studied by direct illumination along the line of specular reflection (D. I. S. R.).

The first group is marked by a varying number of irregularly shaped black pigment specks which impinge on the endothelium as the result of uveal inflammation. The cells themselves are not clearly outlined, and in many instances, the endothelial mosaic assumes a more or less homogeneous appearance, in which only the faintest details of the cell outlines can be seen. The endothelial surface, as a rule, is even. These peculiarities can be seen in almost any part of the endothelium from the pupillary center to the periphery. In the latter location, an occasional Hassall-Henle wart may be observed. This, however, is not a part of the inflammatory process. Vacuoles are rarely found. The changes may be unilateral or bilateral, as the iritis may be unilateral or bilateral.

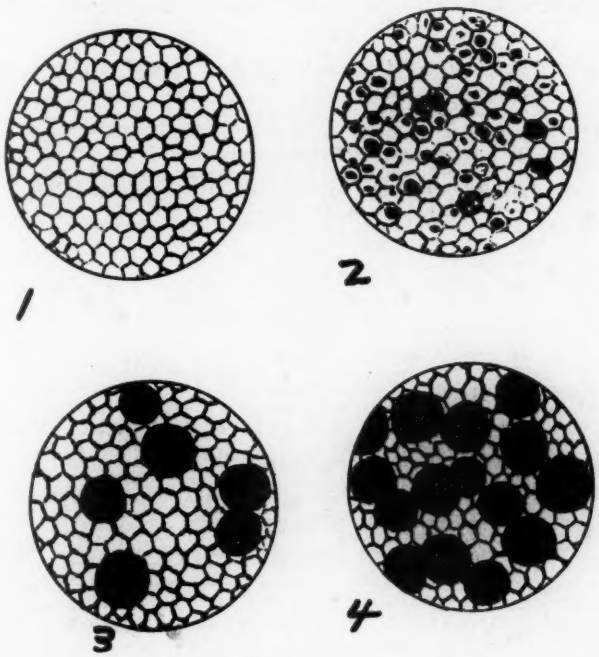
The second group, which is the result of direct trauma to the cornea from foreign bodies, chemical irritations, compressed air explosions, etc., is a unilateral condition, in which the endothelial pathologic process is largely limited to the neighborhood of the trauma. In this group the endothelium shows inequality in the level of the endothelial mosaic. Edema is marked, and appears especially in the form of small, non-reflecting, black spots, which are evanescent and which rapidly disappear with the improvement of the corneal injury. As a rule, the endothelial cells themselves retain a very definite hexagonal outline, and in this respect differ markedly from the changes found in uveal inflammation. Furthermore, in a majority of instances, the epithelial surface of the cornea is also involved. If extensive, the epithelial disturbance often prevents a proper view of the endothelial mosaic. The condition is found in the neighborhood of the trauma. The prognosis is very good, and complete recovery is the rule if the corneal injury is not too grave.

The third or last group is that of genuine dystrophy with which this communication is especially concerned. In a paper that has not as yet been published, but that I have had the opportunity of reading, the author was disposed to include traumatic disturbances of the endothelium in the group of genuine dystrophy. The former, however, is unilateral, rapidly clears, and the vacuoles are smaller and less well defined. The endothelial mosaic presents an uneven surface, which is probably due to the edema, and is always monolateral. In endothelial dystrophy about to be described, the condition is permanent, is bilateral in all instances, begins at the center of the cornea and progresses by extension toward the periphery.

Graves recognized four stages in the development of the dystrophy, probably an overrefinement, but recognizable in different cases. His classification might easily be reduced to: first, incipient dystrophy; second, a well established phase, and finally, complete involvement of the entire endothelial surface of the cornea. The second and third stages

are recognized clinically by a + 20.00 diopter sphere before the eye in a luminous or reflecting ophthalmoscope.

The earliest evidence of the process is found in the center of the cornea and can be recognized only by the slit-lamp with meticulous care and refined technic. With indirect illumination, glints are visible on the posterior surface of the cornea. These glints have a golden hue, brownish gold according to Graves. By direct illumination along the line of specular reflection (D. I. S. R.), the glints are readily distinguished from pigment deposits by the appearance of large, round, black nonreflecting areas in the midst of the perfect hexagonal mosaic



Diagrammatic sketches of endothelial changes: *1,* normal endothelium; *2,* endothelial disturbance due to uveitis; *3,* incipieint dystrophy, and *4,* fairly well advanced, but not the last stage of dystrophy.

of the endothelium. These areas are large and round as compared with the irregular black specks which can be seen on the mosaic, on which pigment specks have impinged as the result of iridocyclitis or uveal inflammation. Most careful adjustment of the beam and of the microscope of high power (3 A objective) will bring this defect into perfect focus. It cannot be recognized by any other means. Vision, in this stage, is perfect unless it is complicated by other disturbance of the media which is not a part of dystrophy. As in the other stages of endothelial dystrophy, the condition is bilateral, but one eye may be in advance of the other.

As the condition advances toward the periphery, the second stage is reached, and almost imperceptibly the entire endothelium becomes involved. In these phases there is added an appearance that Graves likened to beaten silver. Normal hexagonal cells become fewer, and the burnished silver network surrounding the black nonreflecting areas can be observed in almost any part of the endothelium. Vision is now reduced, and one finds it difficult to obtain a clear view of the fundus details. When the vacuoles are present in sufficient number to produce the burnished silver effect, the dystrophy is recognized with an ophthalmoscope and a + 20,00 lens. It is difficult to describe the effect when the ophthalmoscope is perfectly focused, but to me it suggests the

Twenty-Two Cases of Dystrophy of the Corneal Endothelium *

	Sex	Age	Refraction†	Additional Clinical Diagnoses
1	F	70	H + AH	Nuclear cataract; toxic choroiditis
2	M	60	M + AM	Nuclear cataract; advanced arteriosclerosis
3	F	55	M + AM	Nuclear cataract; low grade uveitis and general lowered resistance
4	F	51	M + AM	Incipient senile cataract
5	F	77	M + AM	Nuclear cataract; gastro-intestinal toxemia
6	F	61	M + AM	Nuclear cataract
7	F	62	H + AH	Incipient senile cataract
8	F	60	H + AH	Incipient senile cataract
9	F	61	H + AH	Nuclear cataract; chronic glaucoma
10	F	64	H + AH	Incipient senile cataract; cardiovascular renal disease
11	M	64	H + AH	Incipient senile cataract
12	F	47	H + AH	Incipient senile cataract
13	F	67	H + AH	Nuclear cataract
14	F	68	M + AM	Incipient senile cataract
15	M	70	H + AH	Incipient senile cataract
16	F	42	H + AH	Toxic form of cataract of adult nuclei; traumatic keratoconjunctivitis, left eye
17	F	54	H + AH	Incipient senile cataract
18	F	69	H + AH	Immature senile cataract
19	F	50	H + AH	Incipient senile cataract
20	M	60	H + AH	Incipient senile cataract
21	F	58	H + AH	Nuclear cataract
22	F	70	H + AH	Incipient senile cataract

* Cases 8, 11, 15, 17, 19 and 21 are incipient; all the others are well advanced.
† H means hyperopia; H + AH, hyperopia with hyperopic astigmatism; M, myopia, and M + AM, myopia with myopic astigmatism.

appearance of burnished beaten copper. One is conscious of round or irregularly oval depressions or irregularities in an otherwise normal red fundus reflex. It is most characteristic and, in my experience, cannot be confused with any other condition. Added to this rather typical red fundus reflex appearance is inability to obtain a clear definition of minute fundus details. After seeing a slit-lamp demonstration and the appearance with a + 20.00 diopter lens in the ophthalmoscope, one cannot fail to recognize the condition even without slit-lamp confirmation. It has been found in my clinic by my youngest assistants, with confidence in the confirmation by the slit-lamp.

Thus far the meager references in the literature have practically been confined to slit-lamp recognition. Little has been said as to its clinical significance and the end-results.

From a study of the cases in the table, in all of which the dystrophy was well advanced, several clinical facts are outstanding:

1. All but four cases occurred in females.

2. The average age of the patients was 61 years. One case developed at 41 and one at 47 years.

3. In fourteen cases there was incipient senile cataract, and in eight cases, nuclear cataract.

4. In two cases, there was central toxic choroiditis.

5. In two cases a low grade uveitis was present, and fine pigment deposits could be made out on the endothelium between the large vacuoles which are characteristic of the dystrophy.

6. In two cases there was advanced generalized arteriosclerosis.

7. Six patients were myopic and sixteen were hyperopic.

8. In one case in which cataract extraction was performed, the operative results were fair but the healing process was long drawn out. The wound was riddled by cystoid blebs, one of which ruptured and required repair.

Without exception the general condition was one of lowered vitality. In each instance, thus far examined, the process was progressive. In a few cases well advanced, few normal endothelial cells could be found.

In confirmation of the degenerative character of the process is found the outstanding clinical coincidence, namely, the presence of either senile or nuclear cataract. In two instances a toxic process was observed, but this was probably in the form of a coincidence rather than an etiologic factor. Another clinical phase, which was present in each case, was that of long impaired health with vascular disease much in evidence. The third clinical phase of interest is that of age. In all but one instance the patients were 51 or more years of age. In the youngest individuals, patients of 42 and 47, there was other evidence of a presenile tendency.

CONCLUSIONS

The impressions that one gets from a study of cases are the following:

1. The condition is progressive and reaches its maximum when the entire endothelium is destroyed.

2. Vision is lowered to about 6/7.5 to 6/9 from this cause alone.

3. It is a degenerative process which, probably, is caused by a lack of nutrition of the ocular tissues.

4. Although it is found in association with senile and nuclear cataract, the two conditions are related only in being an expression of ocular complications in patients suffering from nutritional disturbance.

5. The condition is of much more frequent occurrence than its general recognition would indicate. The cases herein reported were found in my practice during the last year or eighteen months.

6. The dystrophy, when well advanced, can be recognized without the aid of the slit-lamp, but with good technic the slit-lamp verifies the diagnosis in a most satisfactory manner.

7. Patients suffering from endothelial dystrophy are poor risks for intra-ocular surgical procedures. The protracted recovery of the one patient on whom I operated would lead me to operate very reluctantly for lens extraction, unless the patient were informed as to the probable operative complications that are apt to be encountered. Delayed and imperfect union could hardly be ascribed to the dystrophy per se but to general lowering of tissue vitality that one finds in patients who are afflicted with this condition. The case in question has led me to believe that in other instances of lowered resistance in which my operative results were similar in character, the same process may have been present but was not recognized. If the recognition of endothelial dystrophy were of no other practical value, this fact alone would serve as a worthwhile reason to uncover its presence. In the modern method of cataract extraction in capsule, one operates much earlier than in former years, because of the more satisfactory results. In the presence of endothelial dystrophy one would, therefore, naturally delay operation until forced to surgical intervention by total visual incapacity or by reason of some complication.

8. Although vision is considerably reduced in the final stage of the disease in the cases thus far examined, the condition did not progress beyond the stage of complete vacuolization. This stage seems to be a stationary one. There is no tendency to regeneration. Dystrophy, therefore, seems to be the logical term to apply to the process.

RETINAL PERIVASCULAR DELINEATION *

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In the latter part of 1925 a series of studies [1] was begun which in the following years [2] disclosed certain phenomena the interpretation [3] of which demanded an understanding of the so-called retinal perivascular space.

A wealth of clinicopathologic citations [3] on this space had accumulated, but there was a dearth of planned investigation. Though experimental physiology had done much to establish well nigh every branch of ophthalmology, the few and inconclusive studies in this particular field had been accepted with less careful scrutiny than was warranted.

In reviewing these purely research efforts, it appeared that in 1867 Prof. Wilhelm His [4] had presented what seemed to be the first experimental evidence of the existence of a retinal perivascular space. The presentation had been made at the July session of the Scientific Association in Basel. This study, secondary to one of like nature on the lymphatic channels of the brain, began by pointing out some of the anatomic similarities of the brain and eye. His then said that his attention had been attracted to the space that appeared about the retinal vessels in hardened sections. The presence of this space had next led him to conduct a series of experiments by means of forced injections. He evidently used freshly enucleated sheep and pig eyes, and first injected into the central vessels, through a minute cannula, berlin blue in water and then alkannin red turpentine under great pressure until the vessels burst. This allowed the blue dye to outline the "lymph spaces" and yet showed the red vessels by contrast.

He also made an injection into the vaginal space by ligating the membranes and nerve behind the globe and then inserting his cannula between the globe and the ligature. This method apparently affected

* Submitted for publication, June 15, 1931.
* From the Research Laboratory of the Brooklyn Eye and Ear Hospital.
* Presented before the American Ophthalmological Society, June 2, 1931.

1. Evans, J. N.: Angioscotometry, Am. J. Ophth. **9**:489 (July 7) 1926.

2. Evans, J. N.: A Contribution to the Study of Angioscotometry, Brit. J. Ophth. **11**:369 (Aug.) 1927; A Scotoma Associated with Strabismus, Am. J. Ophth. **12**:194 (March) 1929.

3. Evans, J. N.: An Interpretation of Defects in the Visual Field, Arch. Ophth. **3**:153 (Feb.) 1930.

4. His, Wilhelm: Lymph Vessels of the Retina, Verhandl. d. Natur. f. Gesellsch., Basel, July, 1865, vol. 4 (Schweighauser Press, 1867).

only the region adjacent to and including the papilla, and represented not perivascular but perhaps perineural spaces. There was evidence that the vitreous was also injected. (A detachment of the retina from the pigment epithelium sometimes resulted.)

The force necessary to produce the delineation made these results seem very unreliable as proof of true anatomic spaces. Even so, he could not be sure of the continuity of the perivascular spaces about the larger vessels on account of the thick adventitia. In conclusion, he said that he regarded the perivascular system as the drainage system of the retina backward through the nerve.

In 1879, Richard Altmann [5] had applied to microscopic preparations the corrosion methods used in gross anatomy. He had succeeded in outlining tissue spaces in the retina of animal and human eyes which he interpreted as "lymph spaces." His method of "impregnation" concerns the present problem. Fresh retina was immersed in a solution made up of 50 per cent olive oil, 25 per cent ether and 25 per cent absolute alcohol. If castor oil was used instead of olive oil, the ether was omitted. This mixture was thoroughly shaken and was capable of taking up small quantities of water. Attraction of the watery elements in the tissue therefore caused the mixture to enter the fluid-containing channels. After from five to eight days, when the tissue had become saturated with the oil, it was immersed in water which, combining with the excess of alcohol and ether, precipitated the fat in the channels. "Hyperosmic acid" was then used to stain the fat. Corrosion was then accomplished by transferring the tissue to dilute or full strength solution of chlorinated potassium.

Altmann's work was of little value because maceration of the cellular elements and violent distortion of the tissues by the concentrated alcohol and ether created artefacts and prevented localization of the stained fat. Moreover, corrosion made it impossible to interpret the origin and relations of the resulting fatty casts.

It was in 1886 that Harold Gifford [6] announced the results of his first series of experiments. He had injected various pigments and organisms into various regions of the eyes of rabbits. In 1892, his second series of studies [7] appeared. Gifford's interest, as he stated, was concentrated on the large lymph spaces, and not on the fine lymph channels. He therefore made no reference to the relation of the pigment

5. Altmann, Richard: The Value of Corrosion in Microscopic Anatomy, Arch. f. mikr. Anat. **16**:471, 1879.

6. Gifford, Harold: Direction of the Lymph Streams in the Eye, Arch. Ophth. **15**:147, 1886.

7. Gifford, Harold: Further Experiments on the Lymph Streams and Lymph Channels of the Eye, Arch. Ophth. **21**:171, 1892.

particles that he occasionally found in the retina to the perivascular region. He made every effort to minimize trauma during the injection.

Nuel and Benoit,[8] in 1900, had injected india ink directly into the vitreous, and also after aspirating some of the vitreous humor. While such severe trauma had probably rendered conclusions unreliable, they stated that in the human eyes the ink could be seen in the "perivascular channels." They concluded that the "retinal perivascular spaces" acted as a posterior drainage route, but that these had but secondary importance except in certain pathologic and glaucomatous states.

Paterson,[9] having performed similar experiments, in 1904 found no instance of the india ink passing the internal limiting membrane.

In 1924, Schneider[10] had met with like failure, though he used the methods of Nuel and Benoit.

One would naturally turn to the work of Schwalbe[11] (1887 and later) for evidence on anything anatomic related to the eye and particularly to anything concerned with retinal vessels, because of his classic writings in Hoffmann's textbook on anatomy and because he was constantly referred to for his descriptions of the perivascular system. In following up his references, however, it became apparent that he accepted the work of His and made no preparations of his own.[12]

Deutschmann[13] (1879) did much to advance the knowledge of the interchange of ocular fluids, but gave no evidence on the perivascular system.

Giving one's attention to the work of Leber,[14] again one would find that his researches fell short of providing evidence on perivascular spaces. The studies of numerous other workers might also be referred to, but always with the conclusion that no evidence of perivascular space delineation was disclosed.

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8. Nuel, J., and Benoit, F.: The Paths of Elimination of the Intra-Ocular Fluid of the Anterior Chamber and the Back of the Eye, Arch. d'opht. **20**:11, 1900.

9. Paterson, J. V.: Some Observations on the Lymph Flow Through the Eyeball in Man and Certain Animals, J. Path. & Bact. **9**:323, 1904.

10. Schneider, C.: Drainage Paths at the Optic Nervehead, Klin. Monatsbl. f. Augenh. **73**:210, 1924.

11. Schwalbe, G. A. (Ludwig): Lymph Channels of the Retina and Vitreous Humor, Gesellsch. d. Wissensch. z. Leipzig, 1872, no. 24, p. 142; Studies on the Optic Nerve, in Hoffmann: Lehrbuch der Anatomie des Menschen, Erlangen, E. Besold, vol. 2, part 3.

12. A review of the work of His was apparently made for Schwalbe by C. Ludwig in 1872 and submitted as a sort of thesis.

13. Deutschmann, R.: Clinical and Experimental Contributions to Resorption of Pathologic Contents of the Anterior Chamber, Arch. f. Ophth. **24**:12, 1878; The Sources of Aqueous Humor in the Eye, ibid. **26**:14, 1880.

14. Leber, T.: On the Ocular Circulation, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, vol. 2, pt. 2, p. 15, 1903.

Carl Behr [15] reported the results of a study in which he made injections into the optic nerve. He presented his observations in 1914 and 1915. Though he pointed out the danger of creating artefacts, he apparently thought that forced injections gave reliable evidence. He thought that pressure on the control manometer as high as 200 mm. of mercury did not produce artefacts because the fluid emerged from the very fine injection needles only drop by drop. He used india ink emulsions and prussian blue mixtures, and made his injections into the eyes of living animals and freshly enucleated human eyes. Some injections were made slowly over a number of hours, using minimum pressures. Some were done under greater pressures, during a few minutes. Behr concluded that his experiments demonstrated not only a perivascular net, but also a system about the nerve fibers and cells. He considered the two as independent of each other. It would seem obvious that the technic as used by Behr was more apt to produce artefacts and false passages than that investigator admitted.

In 1914, Weed and Wegefarth [16] reported the satisfactory delineation of "perivascular spaces" by the use of a mixture of potassium ferrocyanide and iron ammonium citrate. The formula was practically isotonic with tissue fluids.

	Gm. or Cc.
Potassium ferrocyanide	0.5
Iron ammonium citrate	0.5
Water	50.0

The animal was exsanguinated after fifteen minutes, according to the suggestion of Mott,[17] and the eye was removed and placed in a solution of 40 per cent formaldehyde containing 1 per cent concentrated hydrochloric acid. It remained in this for twenty-four hours. Windows were then cut in the sclera, and the eye was placed in running tap water, after which it was placed in 40 per cent formaldehyde for twenty-four hours. Alcohol dehydration was then started. As hardening of the tissue went on, the hydrochloric acid combined with the ammonium radical, and permitted the formation of the prussian blue particles, the process being so slow as to allow thorough saturation of the entire tissue.

In summarizing the results of these studies, one concludes that the method of His does not seem acceptable as it did not produce results in the living eye. In dead eyes it probably only demonstrated the presence of cleavage planes.

15. Behr, Carl: The Parenchymatous Flow of Lymph in the Optic Nerve and Retina, Arch. f. Ophth. **89**:272, 1914 and 1915.

16. Weed, H., and Wegefarth, P.: Studies on Cerebro-Spinal Fluid, J. M. Research **31**:1 (Sept.) 1914.

17. Mott (Physiology of the Cerebro-Spinal Fluid, Lancet **2**:79 [July 10] 1910) advocated killing the animal by exsanguination because by that means the perivascular spaces of the cerebral tissue were dilated as the vessels emptied.

The procedure used by Altmann did not seem acceptable as maceration of cellular elements and distortion of tissue by concentrated ether-alcohol and corrosion prevented localization of channels. The technic, moreover, favored the formation of intravascular casts rather than the formation of lymph space casts.

The method of Nuel and Benoit did not seem acceptable: first, because it was apparently not consistently reproducible, and secondly, because cellular activity obscured the mechanism of ocular fluid flow.

The technic as advocated by Wegefarth apparently gave satisfactory results in his hands and under conditions closely simulating the normal. His drawing of perivascular delineation is, however, far from convincing.

With this scanty background of past efforts as a starting point, a series of experiments has been performed, the primary aim of which has been the delineation of the retinal perivascular region. Certain preliminary studies were necessary to gain familiarity with the conditions under which the ultimate investigations were made.

EXPERIMENTAL WORK

Certain general principles for the conduct of the experiments were adopted. These may be enumerated thus:

1. Only living eyes were used.
2. Rigid methods of control were used whenever possible.
3. Every effort was made to avoid disturbances of the natural pressure and biologic equilibria.
4. The technic of earlier workers was checked for leads to better methods.
5. Only constant and reproducible evidence was accepted.
6. Certain preliminary dissections and injections in dead eyes were employed to gain familiarity with the conditions of the work.
7. Whenever possible, only adult albino rabbits were used.
8. When injections into the vitreous were made, only the smallest practical volume of the agent was employed.
9. The finest needles procurable were used for the injections.
10. Local anesthetics (cocaine and butyn) were used when possible.
11. General anesthetics were sodium amytal (intraperitoneally), ether or chloroform.
12. Careful fundus and slit-lamp (hand) studies were a part of the preliminary examination to eliminate the possible chance of coincidental disease or anomaly.
13. Injections into the vitreous were made under ophthalmoscopic observation.
14. The Sauter tonometer was used in the earlier studies to estimate the pressure variations. It has a foot-plate, 1 mm. in diameter, and exerts its pressure through a spring. The reading is taken when corneal dimpling is evident adjacent to the foot-plate. Tension was estimated before and after injection,

and it was found that when proper precautions had been observed, the pressure variation before and after injection was negligible.

15. India ink for injections was prepared as follows: The "full strength" ink was first boiled for twenty minutes, then greatly diluted and filtered through eleven layers of coarse filter paper. The filtrate was centrifugated and the opalescent supernatant fluid drawn off and recentrifugated until clear. The precipitate was collected and suspended by adding equal parts of tap water and hexylresorcinol solution S.T.37 (Sharp and Dohme), 1:1,000.[18] This ingredient was added because of its remarkable property of reducing the surface tension of structures that it meets and also because of its bactericidal activity. These features decreased the risk of infecting the vitreous and increased the rate of diffusion of the suspended particles. (It is well tolerated by the vitreous.) The particles in the final preparation showed brownian movement.

16. The ocular pigment preparation for injection was as follows: The eyes of three brown rabbits (six eyes) were enucleated ten days beforehand, windows were cut in them, and they were kept in 1 per cent phenol aqueous solution until used. The pigmented tissues were then stripped away and ground in a mortar with 3 ounces (88.7 Gm.) of distilled water. This was kept at 43 F. over night; then the supernatant liquid that contained the pigment granules was pipetted off and centrifugated. The pigment was then taken up from the bottom of the centrifuge tube and examined under the microscope. The particles were a very pale purple and showed lively brownian movement. The upper layers of the liquid in the tube were pipetted off. This was boiled three minutes in the same tube and no coagulum formed. It was recentrifugated in the same tube. Equal parts of hexylresorcinol solution S.T.37 and the sediment were mixed, and the syringe was then charged with the mixture. This was then injected into the vitreous.

17. The preparation of carmine granules for injection into the vitreous was as follows: A small quantity of carmine was mixed thoroughly with hexylresorcinol solution S.T.37 by shaking. This mixture was allowed to stand over night and the supernatant fluid was pipetted into a centrifuge tube. This was centrifugated for fifteen minutes at about 3,000 revolutions, and the supernatant fluid was again pipetted off. The remaining precipitate when examined under the microscope showed very minute particles of the carmine which were apparently of uniform size and showed active brownian movement. Without further dilution this was injected into the vitreous.

The following types of experimentation were performed:

Preliminary Studies: 1. Gross dissections were performed on the freshly enucleated eyes of sheep, dogs, cats, rabbits and guinea-pigs (approximately fifty eyes).

2. Vitreous dissections and injections were performed on freshly enucleated sheep eyes after the technic of Redslob.[19] Slit-lamp studies were made, and injections into the anterior vitreous as performed by scleral and central corneolenticular puncture were studied. Injections of the Wegefarth solution and of the various particulate stains mentioned were made into the vitreous both before and after dissection (about one hundred eyes).

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18. Feirer and Leonard: The Bactericidal Activity of Hexylresorcinol in Glycerine, Bull. Johns Hopkins Hosp. **41**:21 (July) 1927.

19. Redslob, B. E.: A Contribution to the Study of the Structure of the Vitreous, Ann. d'ocul. **164**:107 (Feb.) 1927.

3. Injections of india ink suspensions were made at various depths in the vitreous of living eyes (six eyes).

4. Injections of carmine suspensions were made at various depths in the vitreous of living eyes (four eyes).

5. In one instance a rubber drain was placed in the cisterna magna of a rabbit and kept draining for nine days. Both eyes had been given injections (vitreous) of carmine nineteen days previously, and one eye was enucleated at the time the drain was inserted. (There was no ophthalmoscopic or microscopic difference noted in the two eyes in the dissemination of the granules.)

6. An attempt was made to block the reticulo-endothelial system by the intraperitoneal injection of boiled trypan blue (four eyes). Another attempt to block

Fig. 1.—The appearance of the fatty casts in the retina as stained with osmic acid. Note the distortion and destruction of the cellular elements and the irregularity of the masses. Altmann technic.

the endothelial cells of the eye was made by injecting boiled trypan blue into the vitreous (four eyes). When apparent saturation of the reticulo-endothelial system had been obtained, the particulate stains (india ink) were again injected into the vitreous, but cellular activity was still very great.

7. India ink was injected into the cisterna magna of the living rabbit, and a weak electrical current was passed in the direction of one eye in an effort to encourage movement of the particles through natural channels (four eyes). A similar current flow was created from an eye in which india ink had been injected toward an electrode placed in the cisterna (two eyes). In neither case could dissemination of the particles be influenced by the currents. Replacing the ink with weak nitrate of silver solution gave no more encouraging results (four

eyes). (In these experiments nonpolarizable electrodes were used, and every effort was made to control deposits.)

8. The "impregnation" method of Altmann was also employed, but met with complete failure as would be expected (two eyes).

9. In attempting to repeat the method of His, the eyes of living rabbits were used (in accordance with my adopted policy) (four eyes). The ophthalmic artery proved to be so fine in these animals as to make injections into it impossible. Injections of 30 cc. of prussian blue into the carotid artery, under the greatest possible hand pressure, failed to rupture retinal vessels, but killed the animal instantly.

10. An effort was also made to inject various fluids into the retina so that the avenues of escape might be studied (four eyes). Detachments could be easily produced (seen ophthalmoscopically), but whether of the retina or the choroid could not be determined. Microscopic examination, however, showed no detachment, and the stain was found only in the vitreous.[20]

It would not be profitable to discuss in detail the types of study enumerated, but in view of so much stress being laid in the past on the significance of particulate stains, a typical study must be cited:

In an albino male rabbit (R.le.) injections were made into the vitreous of the right eye; the left served as a control.

March 13, 1928: Both pupils were dilated with 1 per cent atropine sulphate, 1 minim (0.06 cc.), and 1 per cent butyn, 1 minim, was injected into the left eye. This procedure was repeated. Macroscopically, there was no evidence of pathologic changes. No abnormal congestion of the conjunctival vessels was seen. On ophthalmoscopic examination, both eyes showed the albinotic type of fundus without evidence of normal or other pigmentation. There was no suggestion of pathologic changes. A lid speculum (human nasal speculum) of the wire type was inserted. Slit-lamp (hand type and large Gullstrand apparatus) examination showed no change of significance.

A minute drop of india ink suspension was injected by hand pressure into the anterior vitreous of the right eye under ophthalmoscopic observation. The needle passed very obliquely through the sclera from before backward.

March 14, 15 and 16: The slit-lamp or ophthalmoscope showed no demonstrable changes.

March 17, 1928, 2:22 p. m.: Right Eye: The bulbar conjunctiva showed slight injection. The iris vessels were full, and the stroma seemed to be a dirty gray as compared with the other eye, which showed no reaction. The slit-lamp, however, failed to show pigment granules. The pupil was active. There was no evidence of iris exudate or deposits on the posterior surface of the cornea, anterior or posterior lens capsule. The anterior chamber showed a visible beam to the slit-lamp, and a few free cells could be definitely seen. The left eye showed an "optically empty aqueous." The lens showed no demonstrable changes. The vitreous showed a definite cloudiness which resolved itself into indistinct grayish points that may have been either exudate or pigment particles or a combination of both. These particles seemed to show fine fibrillations in relation with them, but there was no suggestion of orderly distribution. These changes seemed to be at varying depths and so obscured fundus structures as to make detailed study

20. A considerable number of other experiments were performed, the outcome of which did not contribute directly to this study.

unsatisfactory. There seemed to be more vitreous opacities immediately adjacent to the ink. The injected ink seemed slightly more widely distributed than previously. Subjectively there seemed to be a suggestion of slight irritation, restlessness and partial closure of the lids.

March 19, 3:45 p. m.: Left Eye: For the first time the left eye showed a marked exophthalmos (estimated at about 3 mm. more than the right eye). There was no change in the external appearance or in the winking reflex. There was, however, no evidence of intra-ocular change. (This exophthalmos was evidently due to the muscular action of which these animals are peculiarly capable.[21]) Right Eye: This eye presented the same appearance as on March 17, except that quite definite channels in the vitreous were outlined and showed bifurcations. The iris and superficial injection was no more marked.

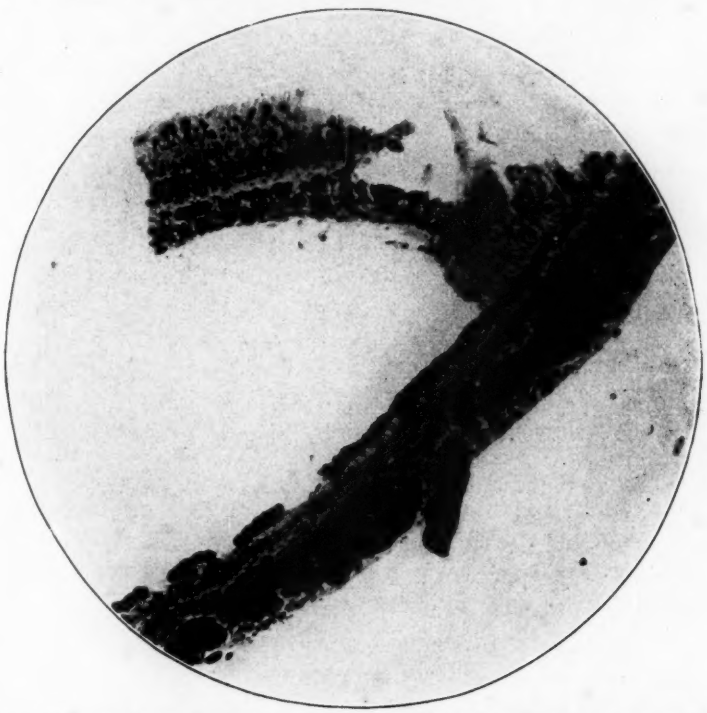

Fig. 2.—Preparation the same as in figure 1, but showing the fat within the choroidal vessels. It is probable that the spaces described by Altmann as casts of lymph channels were casts of blood vessels and casts of crevices caused by violent shrinking. Altmann technic.

March 20 to April 3: Right Eye: This eye showed progressive cloudiness of the vitreous and external evidence of irritation. The iris was becoming darker, and the injected pigment was scattering slowly through the vitreous. Left Eye: There was gradually more exophthalmos but no evidence of pain or inflammation. No evidence of pigment was seen in the fundus.

April 18: Right Eye: The large mass of pigment was still present at the site of injection, but there were also irregular round masses scattered throughout the

21. Pragen, A.: A Study of the Comparative Anatomy of the Extra-Ocular Muscles, Tr. Am. Ophth. Soc., 1928, p. 353.

vitreous and also apparently in and over the retina. There were masses definitely before and apparently on the nerve head. The iris showed a dirty gray color; this may have been due to a reflection from the pigment or to a very fine pigment in the meshes. The rabbit was killed with chloroform; both eyes were enucleated and placed in 5 per cent formaldehyde for hardening. Windows were cut twelve hours later. Left Eye: This eye was consistently normal on repeated examination.

The gross changes in these particulate stain studies may be summarized by a single description, as whatever granules were used, the course was essentially the same. The india ink tended to scatter more rapidly. The carmine scattered apparently less rapidly and tended to be walled off. This might have been more apparent than real, as any phagocytic cells would be less apparent when containing the semitransparent carmine granules than when containing the black ink particles. In cases in which approximately more than 2 minims (0.18 Gm.) of the substance was injected, the eyes tended to develop phthisis as if the injection set up a severe reaction. (It must be recalled that the vitreous of the rabbit is probably more tolerant of abuse than that of the higher animals.)

When the particulate stains were first injected—for instance, in the anterior vitreous, i.e., in the neighborhood of the equator and well toward the peripheral end of the meridian (near the retina)—the first forty-eight hours showed a simple globular mass at the site of the injection. The picture was not materially different when the injection was made at the posterior pole of the lens. At the end of this time, or soon after, there was a prickle-like appearance observable over the surface of the mass, and in a day or two, definite radiations, like streamers, appeared. These were most pronounced, proceeding from the lower edge of the pigment mass. Careful focusing of the ophthalmoscope or slit-lamp showed that the pigment seemed to be in fairly large clumps. There was a tendency for the streaks to split, and in some instances they definitely showed bifurcations.

At the end of two weeks [22] or thereabouts, the pigment masses were seen against the retina, mainly below, though some were more scattered laterally and at varying depths through the vitreous. Those lying on the retina gradually showed fine streaklike edges suggestive of the bone corpuscles of retinitis pigmentosa, but of a more delicate structure.

During the next two weeks the pigment masses seemed to cluster more and more over the region of the nerve head.

This course and disposition seem to be characteristic of carmine, india ink and ocular pigment.

22. In the various experiments, various times were allowed between injection and enucleation.

The microscopic examination of sections from these studies may also be massed for survey, as each pigment followed the same form of dissemination as the others.

MICROSCOPIC EVIDENCE OF THE DISSEMINATION OF THE PIGMENT

The most significant feature of the microscopic study was that the granules of india ink were all taken up by the histiocytes (large mononuclear tissue cells) and were to be found in the tissues very broadly scattered and apparently without constant relation to any hypothetic

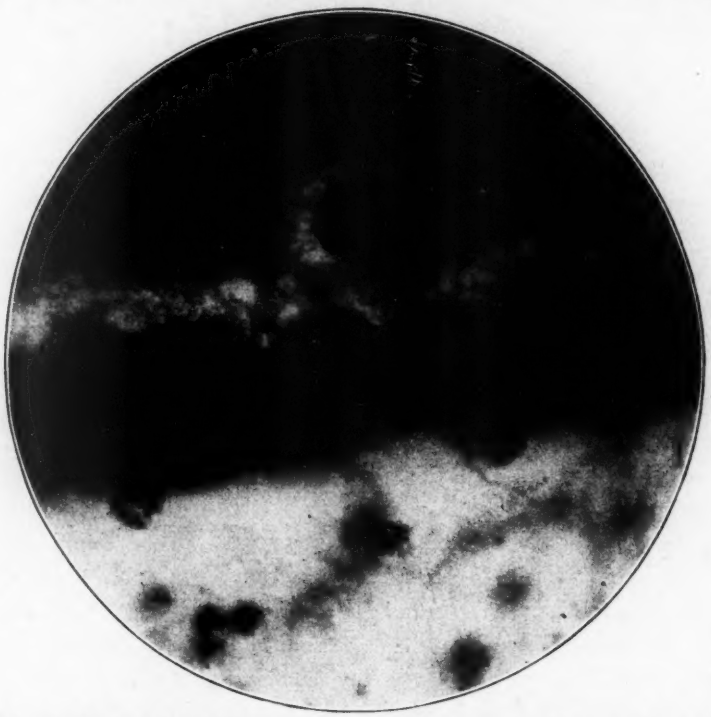

Fig. 3.—India ink injections: There was no natural pigment in the rabbit's eye (albino). The india ink particles are seen within the large wandering cells (histiocytes). These cells pass into the various structures without regard for fluid flow or known channels. Great numbers migrate into the substance of the nerve head where they are scattered about irregularly, singly and in clusters.

or actual spaces or channels, and none were actually seen within the lumina of vessels.[23]

The vitreous showed irregular stringlike arrangements of the cells as well as isolated areas of grouping.

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23. The iris stroma also showed the cells. The region of the suspensary ligament showed a grouping as if the cells had met some sort of limiting membrane in passing forward from the vitreous and were clustered over its surface in the process of working their way through.

Many cells were arranged in rows and clumps at the junction of the vitreous and retina, but the most noteworthy feature seemed to be the fact that the greater number of cells was scattered through the fiber layer of the retina, seldom being found in deeper layers. They showed no greater tendency to cluster adjacent to vessels than elsewhere, but occasional cells were in very close contact with their walls.

The most spectacular feature of the whole picture was the large number of pigment-bearing cells found within the substance of the optic nerve. These cells were visible in all sections of the nerve as far back as nerve tissue was shown. Here also no definite relation could be made to any particular tissues or channels, with the possible exception that more cells were noted in the region about the central vessels in the nerve.[24]

A few cells were found in the choroid but none in the sclera.

The general inference from the studies as outlined is that particulate staining is not adaptable to a study of lymph flow in the sense of dissemination by stream flow, because the tissue cells pick up particles and apparently wander through certain retinal structures without reference to hypothetic or actual channels. If this is a correct view, particulate staining is unsatisfactory for perivascular space outlining.

The use of solutions instead of suspended particles was an early suggestion as more nearly simulating the natural conditions.

It is obvious that all dyes [25] are not appropriate for injection into the vitreous as indicators to show the direction and limits of current flow. Duke-Elder [26] has pointed out that certain of these with large molecules, such as trypan blue and congo red, will not enter the eye from the blood. If positively charged cations are added to the blood, such as the basic dyes—neutral red, methylthionine chloride U. S. P. (methylene blue), cresyl blue and fuchsin—they will not pass the capillary walls. The same holds for metallic ions, such as sodium, mercury and bismuth. On the other hand, negatively charged anions enter readily, such as dyes like eosin, bromides and organic anoidal substances, such as salicylic acid. This would lead one to believe that

24. The length of time that the pigment remains in the eye does not materially alter the picture. There is no apparent scattering until the histiocytes appear. At the end of eight weeks there was still no evidence of lymphatic channel outlines. Perhaps larger injections would have shown earlier scattering, but such technic departs too widely from the natural conditions.

25. Zeigler, M., and Mendel, L.: The Transportation and Elimination of Organic Dyes by the Animal Organism, Am. J. Physiol. **82**:309 (Oct.) 1927. Woollard, H.: Recent Advances in Anatomy, Philadelphia, P. Blakiston's Son & Company, 1927, p. 113.

26. Duke-Elder, W. S.: Clinical Application of the Newer Conceptions in the Pathology of the Eye, Lancet **1**:6 (Jan. 4) 1930.

the first group would not pass from within the eye through the vessel wall, whereas the second group would. Methylthionine chloride, fuchsin, etc., will stain tissues so diffusely, however, that fluid flow is not indicated by their distribution.

As suggested in recording the earlier studies, india ink was one of the most popular of the particulate stains because of its obvious inertness and the ease with which it could be followed in its travels through the tissues. The coarseness of its particles—particularly in the less refined preparations—and the fact that it could be easily con-

Fig. 4.—A section taken through the retina so near the nerve head that the fibers are the predominating structure seen. The larger vessels are shown cut in cross-section, obliquely and lengthwise. The black mass is disorganized blood and is not ocular pigment as the animal was an albino. Weed-Wegefarth-Mott technic.

fused with natural pigments limited its usefulness. Prussian blue was therefore introduced at quite an early date. According to Gifford,[27] Memorsky was evidently the first to suggest this agent. He introduced a solution of potassium ferrocyanide into the eye, and by placing the globe in ferric chloride, created the blue precipitate. Knies, Weiss and Ulrich[28] likewise used this method and evolved elaborate theories

27. Gifford (footnote 7, p. 172).

28. Knies, Weiss and Ulrich, quoted by Gifford (footnote 7).

based on the presence of certain lines and streaks found in the eyes so prepared. Gifford repeated this study and got very similar results, but he was soon forced to conclude that the deposits had no relation to current flow, but occurred wherever the reagents chanced to meet.

Other methods of precipitating these prussian blue particles might suggest themselves. The most encouraging was the technic suggested by the optimistic reports of Weed and Wegefarth (fourteen eyes). It will be recalled that the formula that Weed and Wegefarth [16] used for their injection work was as follows:

	Gm. or Cc.
Potassium ferrocyanide	0.5
Iron ammonium citrate	0.5
Water [29]	50.0

Wegefarth's technic was applied as follows:

The animal was killed by exsanguination fifteen minutes after injection, according to the suggestion of Mott. The eye was then removed and placed in a solution of 40 per cent formaldehyde containing 1 per cent concentrated hydrochloric acid. It remained in this for twenty-four hours. Windows were then cut in the sclera, and the eye was placed in running tap water, after which it was placed in 40 per cent formaldehyde for twenty-four hours. Alcohol dehydration was then started.

As hardening of the tissue went on, the hydrochloric acid combined with the ammonium radical [30] and permitted the formation of the prussian blue particles. Penetration of the tissue was rapid and uniform. Mott advocated killing the animal by exsanguination because by that means the perivascular spaces of the cerebral tissue were enlarged as the vessels emptied.

By using the same methods of control and precautions as already outlined, very consistent results have been attained. These results are uniform when the technic is carefully adhered to. The advantages of the Weed and Wegefarth solution, as pointed out by them, are: (1) that prussian blue particles are easily precipitated by the method advocated; (2) that the blue particles are easily identified microscopically; (3) that the precipitate is insoluble in the reagents used in making and staining the sections; (4) that the reagents are not taken up by living cells; (5) that the reagents used are not toxic when properly employed; (6) that the injected reagents will not diffuse through

29. This aqueous solution is practically isotonic with tissue fluids and is made up fresh just before injection.

30. The solutions must be freshly prepared from the salts as the ammonium radical soon escapes from the solution, and attempts to replace it by the addition of ammonium hydroxide give unsatisfactory preparations.

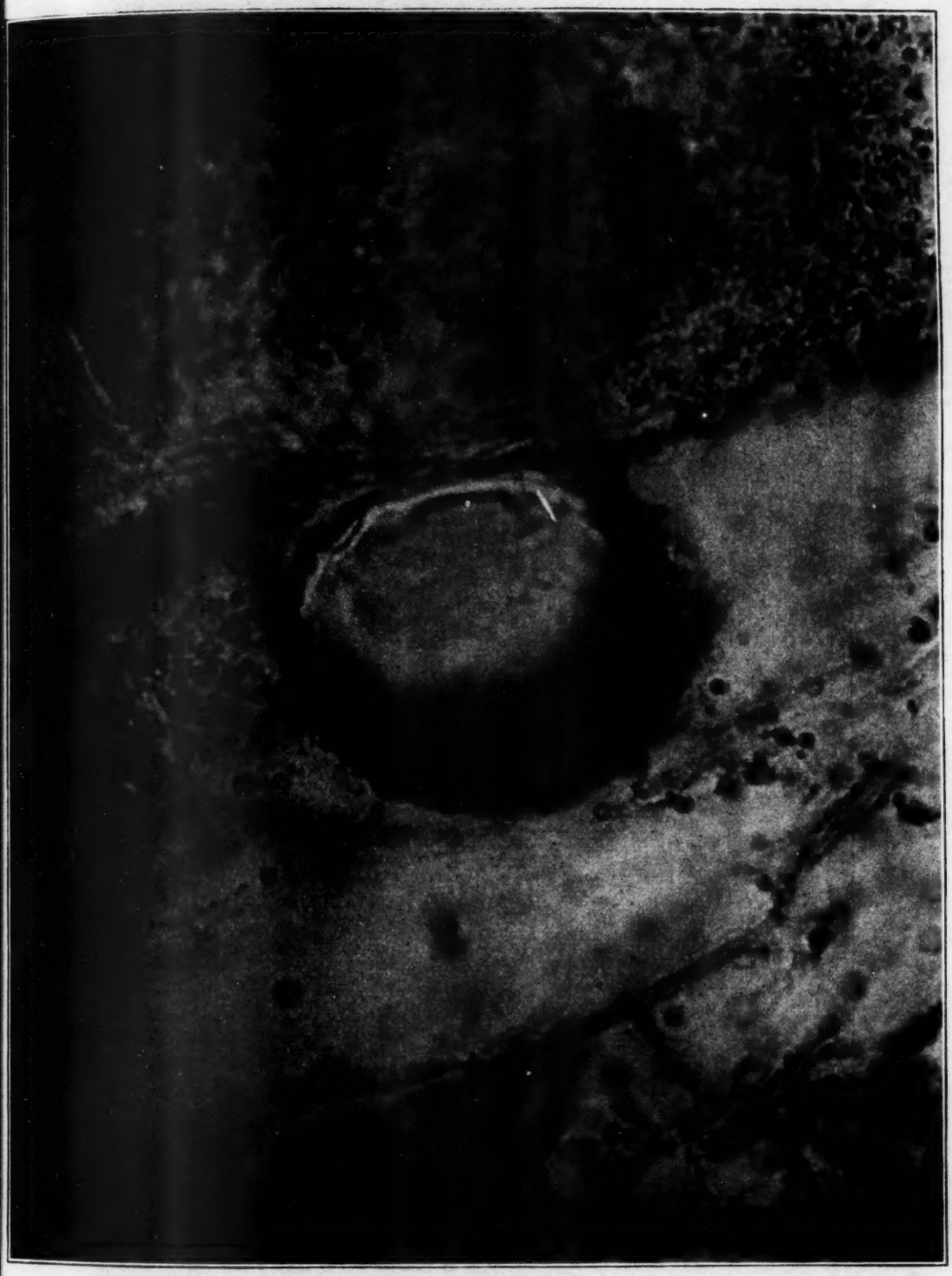

Fig. 5.—A greater magnification of figure 4, showing the cross-cut vessel lying on the obliquely cut vessel. The nuclei of the endothelium are distinctly seen. The granules of prussian blue are in focus in places and out of focus at others. None are seen within the vessel. These sections were stained very lightly with eosin only. Weed-Wegefarth-Mott technic.

Fig. 6.—A large ganglion cell is seen enveloped in prussian blue particles. No nuclear stain was used, and the cell body is visible by virtue of the prussian blue. With the binocular microscope other particles could be seen along the fiber leaving the cell. An occasional bipolar cell may be seen faintly coated with granules. Weed-Wegefarth-Mott technic.

adjacent tissues, and (*7*) that the reagents can be used in solutions isotonic with body fluids.

The accompanying illustrations demonstrate the evidence disclosed by this particular method when applied to rabbits' eyes. The evidence may be summarized thus:

Under the conditions of these experiments: 1. Granules of prussian blue are found about the bipolar cells and about the ascending and descending fibers of the bipolar cells.

2. Granules of prussian blue are found about the ganglion cells and about their fibers.

3. Granules of prussian blue are found about vessels of the retina.

4. Granules of prussian blue are found about the central vessels of the optic nerve.

From this evidence the conclusion is drawn that under the conditions of these experiments the isotonic solution, when injected into the vitreous under conditions closely simulating the normal, penetrates the pericellular and perivascular regions.

A microscopic section of a traumatized human eye showed a heavy perivascular deposit of blood pigment so similar to the sections of this experimental study that application of the methods herein used seem worth while. Six human eyes have already been subjected to the technic with somewhat encouraging results, but appropriate material is so difficult to obtain (negligible pathologic changes) that this final step of the investigation must be left until a later time.

One must be careful in deductions as to the significance of the evidence presented by this study. One can state only that the prussian blue particles are located about the vessels and cells, but must not assume that they are in a formed space, in an anatomic sense, and one must not assume that their passage from vitreous to retinal and nerve spaces indicates the rate or direction of normal fluid flow.[31] I must emphasize that death by exsanguination insured the most uniform results, but there is evidence to show that this measure is not an absolute necessity.

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31. Priestley-Smith is quoted by Weed and Wegefarth (footnote 16, p. 138) to the effect that direct measurements of aqueous drainage show that one fiftieth of the fluid leaves the eye by the posterior pathway.

POSTOPERATIVE NONEXPULSIVE SUBCHOROIDAL HEMORRHAGE *

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Bleeding under the choroid may occur in connection with injuries, operations or perforating ulcers of the cornea. This contribution is restricted to cases in which operation has been performed and in which the hemorrhage has remained confined to the perichoroidal space. In the so-called expulsive hemorrhage, the bleeding takes place with such force that the choroid is ruptured and the blood in flowing out of the opening in the globe carries with it most of the intra-ocular contents. In the form here considered, the choroid is not torn.

REPORT OF CASES

CASE 1.—A woman, aged 65, had glaucoma. The cornea was steamy. The pupil was contracted by miotics to the size of a pinpoint. The anterior chamber was very shallow. Vision was 6/36. Iridectomy was performed. Immediately after the excision of the iris, lens matter presented in the wound, and the patient was seized with excruciating pain. The next day the anterior chamber was found to be filled with blood, and from the gaping wound a mass of bloody vitreous protruded. One month later, the eye was enucleated because of pain and blindness.

Anatomic examination revealed that the capsule of the lens was present, but the substance of the lens had been extruded. An extensive hemorrhage was present under the choroid, enveloped in a capsule of connective tissue.

CASE 2.—A woman, aged 78, had inflammatory glaucoma. The fellow eye had previously been removed following an operation for glaucoma. The anterior chamber was shallow. The iris was atrophic. The optic nerve was completely excavated. While an iridectomy was being performed, lens matter, with a bead of vitreous, presented in the wound. After six days the patient left the hospital, although some vitreous was still protruding. Four weeks from the date of operation, the eye was removed on account of iridocyclitis.

Anatomic examination revealed the iris and the capsule of the lens in the wound. There was a large subchoroidal hemorrhage surrounded by a membrane of connective tissue.

CASE 3.—A man, aged 53, had absolute glaucoma, which had started three months before with an acute onset. The cornea was hazy, the anterior chamber shallow and the pupil wide. Tension was plus 2. The patient had amaurosis. An iridectomy was complicated by loss of vitreous. Eighteen days after the operation, the eye was enucleated because of severe pain that could not be relieved.

Anatomic examination revealed that the iris and ciliary body were caught in the wound. There was a shallow hemorrhage under the choroid, confined to the equatorial region. The blood was not encapsulated.

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* Submitted for publication, March 18, 1931.

* Read before the Section on Ophthalmology at the Eighty-Second Annual Session of the American Medical Association, Philadelphia, June 12, 1931.

CASE 4.—A man, aged 68, had chronic simple glaucoma. The other eye had previously been operated on for glaucoma. The remaining eye was painful and blind. At the end of an iridectomy, vitreous presented in the wound. Twenty days later, because of intense pain, the eye was excised.

Anatomic examination revealed the lens intact and apparently not dislocated. A large hemorrhage occupied the perichoroidal space, partially surrounded by a fibrous capsule.

CASE 5.—A man, aged 65, with absolute inflammatory glaucoma, stated that vision had been normal until four weeks before he presented himself. There was ciliary injection; the cornea was steamy and the anterior chamber exceedingly shallow. Tension was plus 3. The patient had amaurosis. Cyclodialysis was done. As the tension was not reduced seven days later, an iridectomy was done.

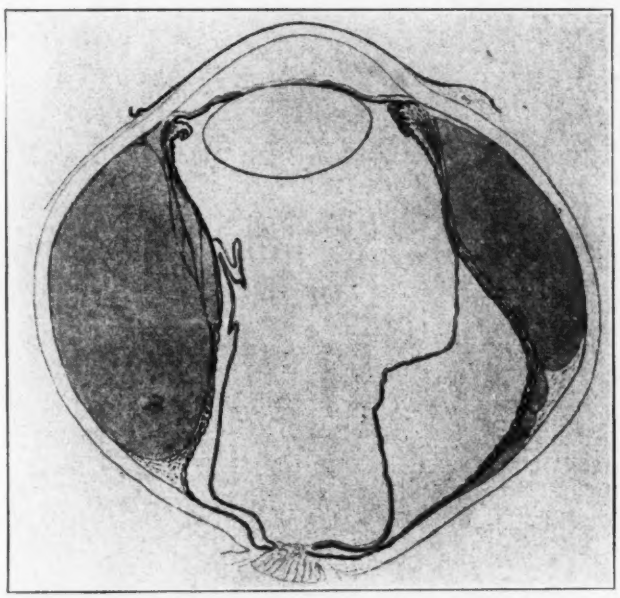

Fig. 1.—Horizontal section: The union between choroid and sclera has been preserved in the posterior segment.

The next day the anterior chamber was filled with blood, and there was some blood on the dressing. Nineteen days after the iridectomy, the eye was removed owing to pain, high tension and blindness.

Anatomic examination revealed extensive subchoroidal hemorrhage. The blood was free in the space, no attempt at encapsulation being visible.

CASE 6.—A boy, aged 6 years, with buphthalmos, presented a steamy cornea. There was a deep anterior chamber. On examination for tension the eye was found to be stone hard. The patient was barely able to perceive light. Elliot's trephining operation was done. Immediately after the excision of the iris, an enormous quantity of vitreous flowed from the wound. The next day the eye was again stone hard and very painful. Fourteen days later it was enucleated.

Anatomic examination revealed the ciliary body caught in the trephine opening (fig. 3). The choroid on the temporal side was pushed almost to the opposite

side by a great mass of blood, so that only a narrow cleft of the vitreous body remained. There was no encapsulation.

CASE 7.—A woman, aged 66, had secondary glaucoma after the extraction of a cataract four months previously. A staphylomatous cicatrix, to which the iris was adherent, occupied the site of the wound. Part of the incarcerated iris was excised. As no reduction in tension resulted, sixteen days later an attempt was made to do an iridectomy inferiorly. Following the incision with a Graefe knife, vitreous escaped. Two days later, the patient suddenly experienced severe pain without any known cause. The eye was found to be hard. Five days after the last operation, the eye was enucleated on account of great pain.

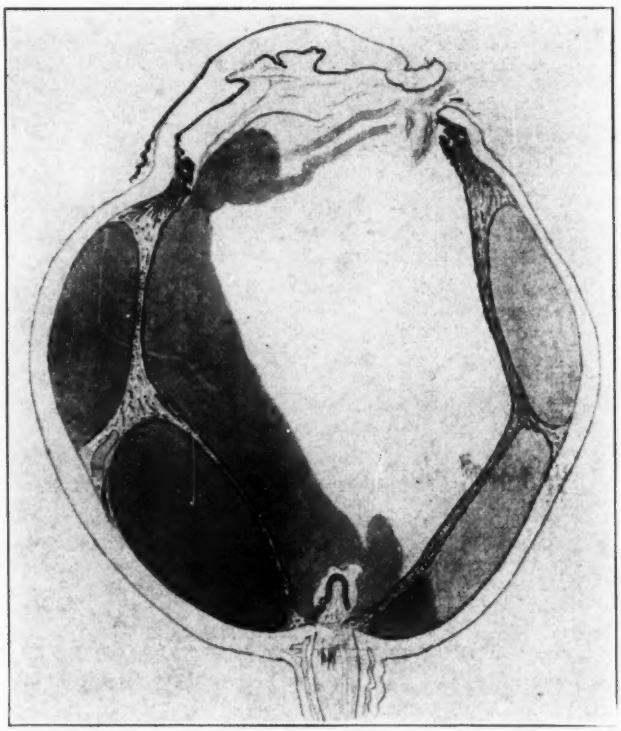

Fig. 2.—Vertical section: Iridectomy for glaucoma. The lens has been extruded. The uvea is completely undermined, and the vortex veins are put on the stretch.

Anatomic examination revealed an enormous hemorrhage under the choroid, not encapsulated.

CASE 8.—A woman, aged 78, had secondary glaucoma after extraction of a cataract. Within a few hours after the operation, the eye suddenly became painful. Iridocyclitis developed, with high tension. Seven weeks after the operation the eye was removed.

Anatomic examination revealed the capsule of the lens in the wound. No lens substance was present. Near the equator, on one side, was a circumscribed thickening of the suprachoroidea, the thickened area being adherent to the choroid and sclera, respectively.

SURGICAL ASPECTS OF THE CASES

Notations of severe pain coming on suddenly at the time of operation or within a few hours after it, with a gaping wound and loss of vitreous, are included in most of the histories. Postoperative high tension is mentioned in each case. These eyes belong to a group with a condition that used to be called glaucoma malignum, in which the tension after operation may be even higher than it was before. In the course of several weeks the wound heals, usually leaving an ectatic scar. The eyes remain hard and painful and become blind, in spite of medicinal

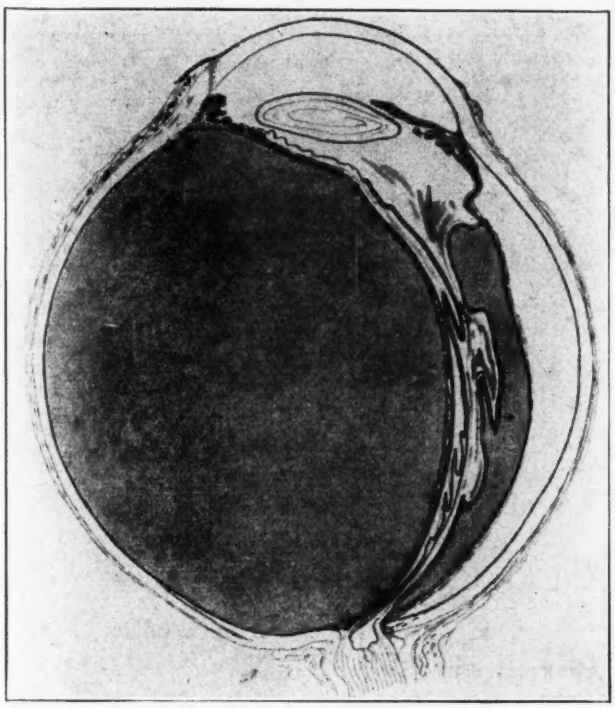

Fig. 3 (case 6).—Vertical section: Massive bleeding with rupture of the vortex veins superiorly. The bleeding ceased only after the ciliary body blocked the trephine opening.

and surgical treatment. The histories and anatomic examinations in the foregoing cases, gathered by chance from various sources, suggest that most, if not all, eyes suffering from glaucoma malignum after operations are lost by reason of bleeding under the choroid. As there is no way of diagnosing the condition positively at the time, it bears the name of "concealed subchoroidal hemorrhage." It is said to occur more frequently in women than in men. The subject is of clinical and pathologic importance.

ANATOMIC CONDITIONS IN NONEXPULSIVE SUBCHOROIDAL HEMORRHAGE

The amount of blood may be just enough to cause a low circumscribed detachment of the uvea far forward, or it may be so large as to undermine the uveal tract on one side, from the scleral roll anteriorly to the margin of the optic nerve. Usually the blood does not undermine beyond the limits of the perichoroidal space. As this comes to an end posteriorly at a considerable distance from the nerve head, it is exceptional to find blood under the choroid in the posterior segment of the eye. The vortex veins superiorly and inferiorly fix the choroid to the sclera, in the

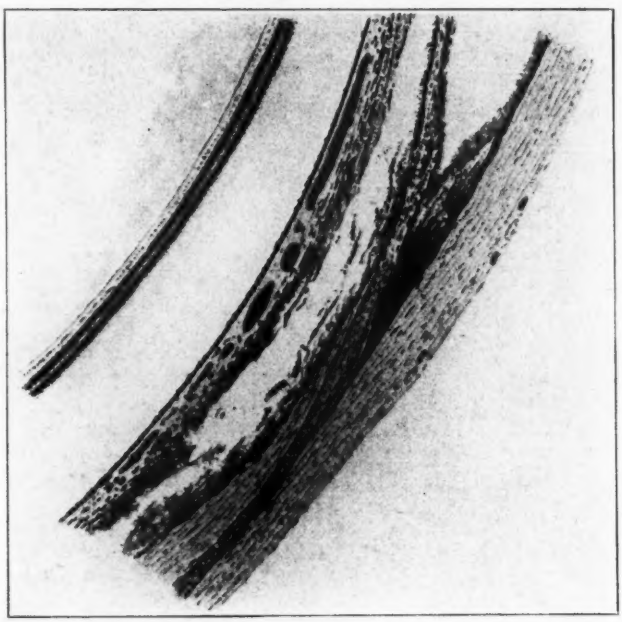

Fig. 4.—Horizontal section: The long posterior ciliary artery is ruptured at its point of entrance in the perichoroidal space. The nerve shows the effects of traction.

region of the equator. For this reason the blood accumulates on the nasal or temporal side, where there is nothing to prevent the choroid from detaching itself from the sclera. Some blood is apt to find its way to the opposite side by stretching the vortex veins. The blood separates into serum and erythrocytes, the latter accumulating at the most dependent part, which corresponds to the position of the head. The retina remains adherent to the choroid in all cases, because both layers are compressed against the vitreous body. The latter in turn pushes the lens and iris forward into the wound. It is noteworthy how often the capsule of the lens ruptures, allowing the substance to escape, while the capsule is caught in the wound. I have in mind an iridectomy for glaucoma in

which the nucleus of the lens was found in the conjunctival sac the morning following the operation, after the patient had suddenly experienced great pain during the night.

HISTOLOGY OF NONEXPULSIVE SUBCHOROIDAL HEMORRHAGE AND THE MANNER OF ITS DISAPPEARANCE

There is seldom any free blood in the stroma of the choroid, which may already be greatly altered in glaucomatous eyes. Owing to the compression of the choroid and retina against the vitreous, areas of

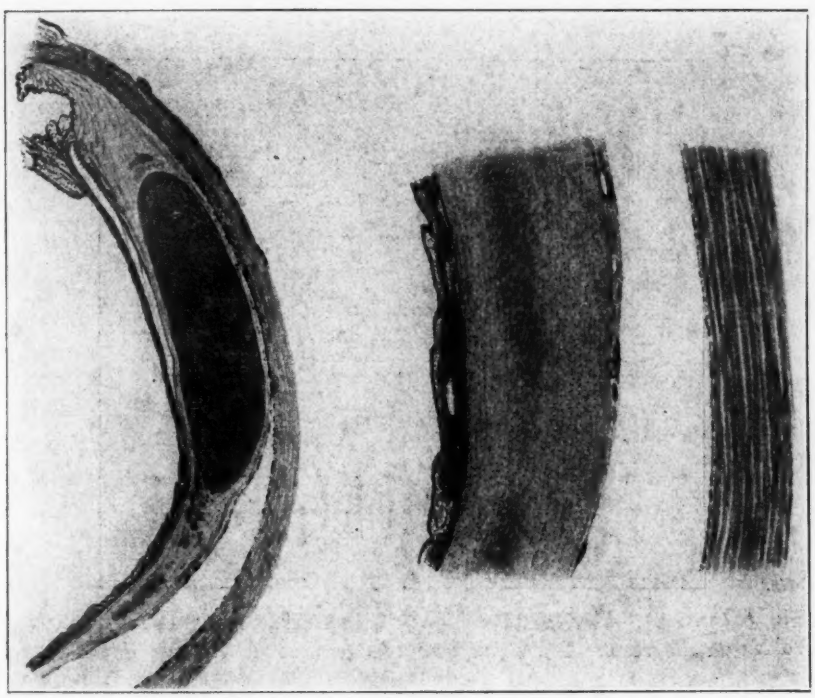

Fig. 5.—Horizontal section: Subchoroidal hemorrhage, completely encapsulated. The detail from the lower angle shows the dense, avascular connective tissue that results from a fusion of the walls on the disappearance of the blood.

actual necrosis may be produced in these layers from a cutting off of the circulation. In case 6 (buphthalmos), in the area where the retina and choroid are pressed against the lens, necrosis is seen, probably due to direct pressure on the tissues. There is always great engorgement of the vessels of the choroid in the posterior segment, where the union with the sclera is maintained . The presence of blood in the perichoroidal space acts as an irritant to the adjacent structures, which react by forming a membrane of connective tissue to envelop the hemorrhage. In cases of extensive hemorrhages in eyes that are probably very hard,

there appears to be a delay in the formation of the capsule. This is demonstrated in cases 5 and 6. In smaller hemorrhages, in which the tension is not apt to be so great, the encapsulation begins earlier and proceeds more rapidly. Usually in an ordinary hemorrhage there is considerable encapsulation within two weeks. As a rule, the capsule is thicker on the choroidal than on the scleral side. The first signs of encapsulation are found posteriorly, where the angle of separation is not so wide as it is anteriorly at the scleral roll. Posteriorly there is a better supply of blood. The encapsulation starts on the choroidal side of the posterior angle, where the short posterior ciliary arteries are exposed by stretching. The capsule increases in thickness at the expense

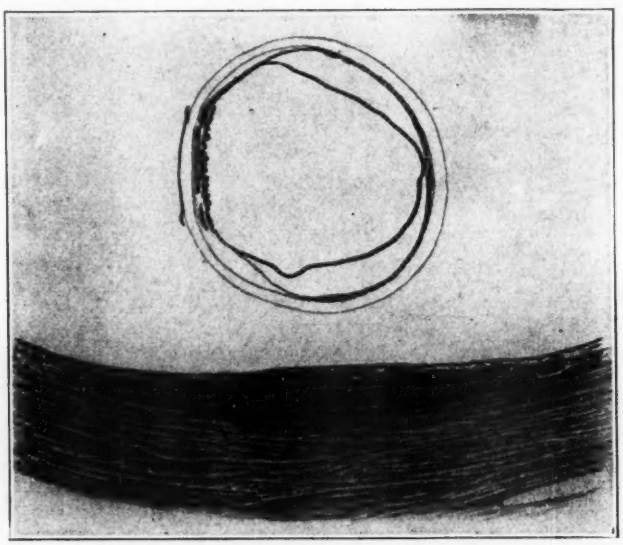

Fig. 6 (case 8).—Horizontal section: Circumscribed thickening of the suprachoroidea as the result of a small hemorrhage (pseudosarcoma of the choroid).

of the enclosed blood. It is composed of thin, elongated fibers running in all directions. It rarely contains blood vessels, and is singularly free from pigment. No prolongations are sent into the hemorrhage, so that the inner surface is remarkably smooth and even. Externally the capsule may be adherent to the choroid and the sclera at the same time, or it may be entirely independent of either, being separated from these layers by compressed subchoroidal lamellae. In a case of sympathetic ophthalmia after an injury (fig. 5), a few strands of the lamellae intervened between the capsule of a large hemorrhage and the sclera. These remnants of the uvea were infiltrated with lymphocytes, showing the affinity that the virus of this disease has for uveal tissue, the presence of so small a residuum being enough to lure the lymphocytes.

In the process of the disappearance of the hemorrhage, phagocytes are rarely encountered, as is common in the periphery of blood clots in the vitreous and aqueous. Nor do the erythrocytes break up into granules. No shadow cells appear. As long as the hemorrhage is present, the red blood cells may show little or no change. They may persist for weeks. The exact mode of their disappearance is not clear. Probably they undergo chemical change, being dissolved, and in this way are carried off by the fluids of the tissues. As the quantity of blood diminishes, the capsule appears to contract or to collapse, so that when the lining finally coalesces merely a thickened membrane is left (fig. 6). In some preparations a row or two of cells, indicate the line of union, and in other instances no sign of fusion is present. In the end, the suprachoroidea is transformed over a greater or lesser extent into a thick, dense, avascular membrane free from pigment. Such thickening of the suprachoroidea is often mistaken for the organization of an exudate after suprachoroiditis, when, as a matter of fact, it represents the collapsed capsule that once surrounded a hemorrhage.

Light is thrown on this matter in the examination of an eye enucleated four months after an injury. The suprachoroidea is replaced over its entire extent, from the scleral roll anteriorly to the vicinity of the optic nerve, by a thick dense membrane. This lies like a ribbon in the sections, completely detached from the choroid and the sclera. Running along its center here and there are lymphocytes and some granules of blood pigment, marking the last vestiges of the hemorrhage.

SOURCE OF THE BLEEDING

The bleeding does not come from the vessels of the choroid; otherwise there would be blood in the stroma. Many of these eyes show small hemorrhages in the retina, but they are of no consequence. It appears that the blood must come from a rupture of one of the two long posterior ciliary arteries. The seat of the rupture is probably at the site of entrance of the artery into the perichoroidal space. In several specimens, fragments of the walls of this vessel were found clinging to the emissarium. In one case the adjacent stroma was infiltrated with blood that had found its way there from the perichoroidal space (fig. 4). The distal walls of the ruptured vessel retract and quickly degenerate in the midst of the blood. As the streaming blood drives the choroid forward, other blood vessels at the anterior and posterior aspects of the perichoroidal space may be torn by traction. The blood continues to escape until the lens or iris, acting as a tampon, plugs the wound, whereupon the intra-ocular pressure begins to rise again until it may be higher than it was before.

CAUSE OF THE BLEEDING

When the intra-ocular vessels are suddenly deprived of the support afforded by the intra-ocular pressure, they dilate and are filled with an abnormal amount of blood. Whether the walls break or not depends on several factors: (*a*) There is a difference in intra-ocular pressure before and after the opening of the globe. Thus, in glaucoma, in which the tension is high, there would be a greater drop of tension than in cases of cataract, in which the tension may be normal. This is one of the reasons that bleeding takes place more frequently in operations for glaucoma than in those for cataract. (*b*) The walls of diseased vessels are more likely to rupture when the intra-ocular pressure is reduced than are those of normal vessels. (*c*) The walls of vessels in glaucomatous eyes are prone to break because the patients are usually elderly persons and because the eyes are likely to be diseased independently owing to the changes brought about by the high tension. (*d*) Certain types of operation, particularly Elliot's trephining, and LaGrange's sclerotomy, are more likely to be complicated by subchoroidal bleeding; this may possibly be explained by the fact that after these operations the tension remains low for a longer time than after ordinary iridectomies. Thus a vessel might be able to stand the complete removal of its accustomed support, the intra-ocular pressure, for a few minutes but not for some hours.

COMPARISON OF THE EXTRAVASATION OF BLOOD WITH THE TRANSUDATION OF SERUM

Extravasation of blood takes place rapidly; the transudation of serum develops slowly. Both are found in greater accumulation forward in the region of the ciliary body. A transudation is confined to the normal limits of the perichoroidal space, while a hemorrhage may undermine the choroid as far as the optic nerve. The two do not occur together. Bleeding, coming on abruptly, leaves no time or space for the transudation of serum. Eyes showing hemorrhage under the choroid are hard; those showing serum are soft. A fibrous thickening of the suprachoroidea persists after the disappearance of blood, but no membrane is found after the absorption of a transudate.

EXPLANATION OF PAIN IN SUBCHOROIDAL HEMORRHAGE AND OF ITS ABSENCE IN TRANSUDATION OF SERUM

In subchoroidal hemorrhage the quick and tense stretching of a long posterior ciliary nerve probably causes the intense pain. As the tension rises to a level as high as it was before, or higher, the pain is kept up from pressure. Doubtless, toxins irritating to the nerve are set free from the mass of blood. Later, when all the blood is gone, if the nerve still

exists it is encased in newly formed tissue and subject to contraction. In transudation, the traction on the nerves comes on gradually, and with the absorption of the fluid the suprachoroidea is again normal.

METHOD OF DESTRUCTION OF THE EYE IN CONCEALED SUBCHOROIDAL HEMORRHAGE

The rupture of one of the long posterior arteries, and in rare instances of both, greatly impairs the supply of blood to the ciliary body and iris, and this may result in necrosis. The degeneration of a long posterior ciliary artery leads to trophic disturbances. In massive hemorrhages the mechanical damage to the tissues may be considerable. The continuance of high tension is of itself destructive. After the absorption of the blood, these eyes become soft and atrophic, with deep anterior chambers.

OPHTHALMOSCOPIC PICTURES

Most of the eyes that reach the laboratory are greatly damaged by subchoroidal bleeding. There are probably many other eyes with milder degrees of bleeding that heal spontaneously, circumscribed areas of thickened suprachoroidea in the periphery of the fundus remaining. The outcome of a small hemorrhage is shown in figure 6. In this instance, as an exception, there are blood vessels and many darkly pigmented cells resembling chromatophores. With the ophthalmoscope the area would probably have appeared as a dark brownish field of some elevation in the choroid. The histologic picture corresponds to that of an eye studied by Meller,[1] in which such an area was diagnosed clinically as melanosarcoma of the choroid, and the eye was removed. It proved to be the result of a small bleeding under the choroid. To this condition the name "pseudosarcoma of the choroid" has been given. It is well to bear in mind the possibility of the existence of such areas in the anterior stretches of the fundi of eyes that have been injured or operated on.

METHOD OF PREVENTION OF SUBCHOROIDAL HEMORRAGE

Since bleeding of any considerable extent under the choroid ends in the loss of the eye, every precaution should be taken to prevent it. This is especially true in persons with glaucoma in whom an operation has not been successful on one eye. Preventive steps should have for their purpose: (*a*) the reduction of the general blood pressure, by such means as dieting, purging, and the administration of hypertonic solutions, and, at the time of operation, general bloodletting, with sedatives; (*b*) the reduction of the intra-ocular tension locally by the use of miotics, in conjunction with epinephrine compounds. Before the opera-

1. Meller: Ueber Pseudosarcoma choroideae, Ztschr. f. Augenh. **49**:235, 1925.

tion, to preclude squeezing, akinesis, as employed by O'Brien,[2] is recommended. It is well to have the head elevated, and, above all else, the aqueous should be slowly evacuated.

TREATMENT OF NONEXPULSIVE SUBCHOROIDAL HEMORRHAGE

In the literature there appears to be but one eye that has been saved after an extensive subchoroidal hemorrhage. This was a case of Verhoeff's,[3] during the performance of sclerostomy for glaucoma. He states that suddenly the vitreous poured rapidly out of the wound, until half of it must have escaped, when abruptly the flow ceased. The eye became extremely hard and painful. Four hours after the operation Verhoeff punctured the sclera to evacuate the blood. He records that in another case he would advise stopping the vitreous flow by digital pressure, making scleral punctures as quickly as possible. He suggests small, triangular incisions, excising an apex of the triangle to afford continuous exit to the blood. In his case, the organ retained considerable vision. Possibly by this method of Verhoeff's, eyes may be saved, even after the blood has been in the perichoroidal space for some days, because, as has been found out, the blood may remain unchanged for an indefinite period and the encapsulation may be long delayed. It is known that the bulk of the blood is apt to be well forward, but there is no way of telling on which side it is. One should puncture the temporal side first, in which location, according to the specimens, the bleeding is more likely to break out and to be present in greater amount.

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DISCUSSION

DR. E. C. ELLETT, Memphis: To establish the condition described, pathologic material is necessary, as the clinical evidence is not sufficient, only suggestive. In searching through specimens from my own cases that have terminated so unfavorably as to require enucleation, I have found only two sections that show the condition under consideration, or something like it.

Neither of these cases fits exactly into the group described in the paper, but they are the only ones in my experience that show a similar condition. We see a good many people who have hemorrhages, usually in the anterior chamber, after cataract extraction, and usually with a history of sudden pain. Whether we can establish the fact of an injury to the eye by the patient's hands or the hands of another, or whether there is a sneeze or cough or other disturbances to explain it, we usually attribute the hemorrhage to an accident, and if the blood clears up without bad effect, we think it came from the iris or conjunctiva. Other patients do not do well, and inflammatory symptoms and rise of tension develop; no doubt the explanation in many of these cases is the condition described in the paper.

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2. O'Brien: Akinesis During Cataract Extraction, Arch. Ophth. **1**:447, 1929.

3. Verhoeff, F. H.: Scleral Puncture for Expulsive Subchoroidal Hemorrhage Following Sclerostomy, Ophth. Rec. **24**:55, 1915.

DR. EDWARD STIEREN, Pittsburgh: I am in accord with Dr. Samuels' suggestions for preventing subchoroidal hemorrhages before operating on eyes with increased tension, except his advice to use epinephrine compounds. The effect of these drugs is to constrict the peripheral vessels, dilate the pupil and reduce tension. This diverts more blood to the deep vessels. An eye in this condition being subjected to sudden evacuation of the aqueous by a corneal section is much more liable to have intra-ocular bleeding.

The last point cannot be overstressed. It is obvious that if the aqueous is permitted to escape slowly, the danger of internal hemorrhage is not nearly so great as when it gushes out, as is usually the case in the large sectioning of the cornea necessary in performing an iridectomy.

Iridotasis is my favorite operation, as the corneal section need not be large and a gradual escape of the acqueous occurs even when the tension is high.

DR. F. H. VERHOEFF, Boston: I am pleased to know that Dr. Samuels approves of the method that I brought forward many years ago for combating postoperative subchoroidal hemorrhage. The patient he mentions came back six years later with acute glaucoma in the other eye. I performed an iridotasis, but the tension remained high, and four days later there was severe pain, and the vitreous poured out of the wound. As soon as I could get to the hospital I tried scleral punctures, but they were not successful; the hemorrhage had ruptured the choroid and ruined the eye. The patient still retained useful vision, however, in the eye that I had been fortunate enough to save.

To prevent expulsive hemorrhage, I do not feel there are many precautions that we can take before operation. We have so few such cases that we cannot ascertain from practical experience what measures may be effective and therefore must rely on theoretical considerations. But the danger of depending on theory is that one is apt to do as much harm as good. For instance, blood-letting preliminary to operation has been suggested, but it seems to me that this might result in a sort of water hammer effect and increase the danger of rupturing a vessel. That epinephrine will be of value I think is doubtful. By contracting the smaller arteries it might lower the vascular pressure in the choroid, but, on the other hand, this might be offset by the increase in the general blood pressure. I think the safest plan is simply to keep the patient in bed, which is what we do anyway.

Expulsive hemorrhages occur so seldom that this condition would be expected almost never to occur in both eyes. However, in addition to the foregoing case, I have had another in which it occurred in one eye and a few years later in the other.

TONOSCOPY *

A. MAGITOT, M.D.
PARIS, FRANCE

The term "tonoscopy" (Terson) designates the method by which one evaluates the blood pressure in the vessels of the retina. This method, devised in 1917 by Bailliart as a clinical means of exploring the ocular and cerebral circulation, has already been fruitful of results and still seems to have a rich future before it.

It is known that the ocular circulatory network, the central vessels, arteries and veins, forms a closed system of the cerebral type, namely, a terminal system without anastomoses. Yet my observations carried out with Bailliart in 1919 have shown that there is a close parallelism between the retinal and uveal circulation, and that the pressure is the same in the central artery at the disk and in the arteries of the iris. This fact has since been confirmed by other observers (Manjukowa and Pietueva, Samojloff).

The retinal artery, arising as it does from the ophthalmic artery, has the same origin as the cerebral arteries which are branches of the carotid. By tonoscopy, at the level of the disk, one has therefore a keyhole through which to view the cerebral circulation, for the pressure must be identical in the retinal artery and in the cerebral arteries of the same caliber.

Following my first researches with Bailliart and the article that I wrote in the *American Journal of Ophthalmology* in 1922 on this new method of clinical investigation, numerous papers have appeared on tonoscopy. In 1927, Kalt was able to collect 130 bibliographic references on this subject. In the last ten years tonoscopy has considerably increased the knowledge of the circulation of the blood not only of the eye but also of the brain, and although still further progress may be hoped for, one may even now gain some idea of its advantages. The aim of the present article is to take bearings and to review what, at present, constitutes the total sum of the knowledge. I wish, in analyzing the facts published and comparing them with my own personal experience, to show the reason for certain discrepancies and demonstrate the value of the method.

Although the technic of tonoscopy is well known, it is best, before proceeding further, to speak of the manner in which the examination should be made in order to give accurate and consistent results.

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First of all, after instilling a few drops of a brand of phenacaine or other suitable drug (butyn, etc.), the ocular tension is taken with the tonometer (Schiötz or Bailliart). Next the systemic blood pressure must be ascertained.

Many physicians take the pressure in the brachial artery with the Riva-Rocci apparatus. This instrument is fitted with a rubber armlet which is blown up with air, and the artery is explored below the compression. With the usual ausculatory method of Korotkow two readings are obtained, one for the diastolic and the other for the systolic pressure. The diastolic pressure is the one that serves as a basis for Bailliart's formula.

This method has, however, a serious drawback, pointed out recently by Vaquez, Gley and Gomez, because it gives no information as to the average arterial pressure, namely, the constant pressure which would insure in the vessels the same flow of blood as results from the existing variable pressure. Now, it is of special interest to the pathologist to know the force by means of which the blood circulates through the organs, and this force is given by neither the systolic nor the diastolic pressure, nor by the arithmetical average of the two figures. The researches of these writers have confirmed those of Pachon in 1922, and have shown that this force could easily be ascertained by the oscillometric method. The instrument (Pachon-Boulitte's sphygmo-oscillometer) is fitted with a rubber compression sleeve, but the exploration of the artery is made at the level of the compression and not below it. When equilibrium is reached between the external pressure and the hydrodynamic pressure within the vessel, the oscillations of the pointer reach their maximum amplitude; that is, the average pressure is reached. In a normal subject having a systolic pressure of 150 mm. of mercury and a diastolic pressure within the vessel, the oscillations of the pointer reach their maxi- to 90 mm.

This method, which consists in estimating the average systemic pressure instead of the diastolic pressure, offers many advantages. Experience has shown that although from one day to another, or during the same day, the diastolic pressure may change, the average pressure varies very little. On the other hand, the method permits of comparison with a similar phenomenon observable on the disk. The compression of the eyeball by a dynamometer is exactly comparable to that exercised on the arm by the pneumatic sleeve. Its effect is to bring about equilibrium between the intra-ocular pressure and the retinal blood pressure. One observes first a preliminary vibration in the artery followed by a steady pulsation when the average retinal pressure has been reached. The first phenomenon is fleeting, whereas the second is very positive. In consequence, the taking of the average arterial pressure facilitates the tonoscopy measurement without changing the Bailliart comparison formula

of 1:2, which states that the pressure in the central artery is about half that in the brachial artery. But as emotion or fear may, in certain patients, modify the blood pressure, it is advisable to take the radial pressure before and after tonoscopy.

A second point of some importance is the manner in which the dynamometer is applied to the eyeball. The convex footplate of the apparatus should be placed on the conjunctiva a little behind the insertion of the rectus externus muscle, and the pressure should be applied from outwards inwards. In order that the compression may be transmitted to the retinal vessels with a minimum dissipation, it is necessary that the eyeball be held firmly against the internal wall of the orbit. Bliedung's method which makes use of a pneumatic cushion applied to the eyelids is incorrect because a part of the compressing force is wasted on the retrobulbar tissues and on the eyelids.

A third point concerns the rapidity with which the compression should be applied. Bailliart and I have drawn attention to the fact that a compression applied too slowly or pressures applied without sufficient intervals between them are liable to cause errors. Any compression of the eyeball, even the repeated application of the tonometer, causes a fall in the ocular tension. This tension should, however, be maintained constant as far as possible; hence the recommendation not to proceed too slowly and to repeat the tonoscopic examination only after a rest of five minutes.

It is advisable to view the artery with the magnification of the direct ophthalmoscope. The observer, holding his ophthalmoscope in one hand, applies the pressure gage himself, with the other hand supported on the temporal region. Some make use of the large or the small Gullstrand ophthalmoscope. It is then necessary for an assistant to apply the compression and note the dynamometer reading at the exact moment when the observer tells him to stop.

Lastly, if the retinal blood pressure appears to be abnormal, it will be wise to repeat the tonoscopy not only the same day but also the next and for several days thereafter. Clinical practice has shown that in certain patients the cerebral circulation is subject to variation. Before assuming the existence of a permanent intracranial hypertension, circumspection is necessary.

THE TONOSCOPIC OBSERVATION

The examination should be made, except in exceptional cases, on the disk at the point of emergence of the vessels. Tonoscopy applies chiefly to the central artery, but studies have also been made on the vein.

Arterial Tonoscopy.—In a healthy adult, unless one makes use of certain high magnifications, the central artery appears to be motionless. Compression of the eyeball raises the ocular tension, and when the latter

begins to equalize the diastolic pressure, the observer sees first a tremor followed by a slight pulsation and then by a steady beat. If one continues to compress the eyeball, the artery is seen to empty and fill itself rhythmically, and finally with increasing pressure it remains completely empty.

The first tremor corresponds to the diastolic pressure, the steady beat to the average pressure and the pulsation just before complete collapse to the systolic pressure. As already mentioned, it is the average pressure which is the easiest to note. It is also the most interesting. The systolic pressure is, clinically speaking, unimportant. It is, moreover, rather difficult to observe because, through the action of a regulating mechanism and in order to overcome the resistance resulting from compression of the capillaries, there occurs an increase in the contractile pulsation of the arteries, the so-called peripheral heart mechanism. The pressure thus ends by being the pressure not of the retinal but of the ophthalmic artery.

The criticism made by Duke-Elder, that this method of using the reading for the average pressure gives no information as to the true figures for the blood pressure in the retinal artery, is justified only if one attributes a value to the absolute figures.[1]

Evidently tonoscopy gives only an approximate value, just as is the case with sphygmomanometry by Riva-Rocci's or Korotkow's apparatus or even by Pachon's oscillometric method. But, leaving aside the fact that Baurmann has shown that the margin of error is not so great as Duke-Elder believes, tonoscopy is only a method of clinical exploration, and all that one requires of the figures is that they should supply constant and consistent values with a given technic and instruments.

Tonoscopy is generally effected with the patient sitting, but it may be carried out also on a patient who is lying down. It must be remembered, however, that although the position of the body has, in most patients, no influence, in certain persons the horizontal position may have an effect on the ocular tension (Thibert, Berens). The retinal pressure should be taken on each eye, for although, as a general rule, the retinal pressure is the same on either side, patients are sometimes met who have an inequality in pressure independent of any objective arterial lesion. These somewhat rare occurrences deserve, moreover, to be carefully sought.

1. The exact figures for the retinal arterial pressure have been much discussed. Certain writers seek by means of a choice of values to justify the filtration theory of the aqueous humor; others, the secretion theory. But apart from the dogmatic side of the question, Duke-Elder considers that what is called the pressure in the radial artery is in reality the brachial pressure, and that what is considered as pressure in the central artery is in reality the pressure in the ophthalmic artery. The proportion between the two being constant, this discussion has no clinical interest.

The pulsatile movements induced are different in children, in adults and in the aged. In childhood and youth the pulsation takes place over the whole surface of the optic disk, not only on the main arterial trunk but also on the branches. It sometimes take the form of veritable jerks. In an adult the pulsation takes place either on the trunk of the artery or on one of the principal branches if the artery divides behind the lamina cribrosa. In the aged the amplitude of the pulsation is much less, the strength of the beat being directly proportional to the elasticity of the walls. Sometimes only a single point of the vessel seems to oscillate.

Among all the characteristics described for the senile retina, certain ones of which are extremely subtle, thinness of the blood stream seems to be the most frequent, according to Bidault, occurring in one third of the cases. Moreover, tonoscopy reveals the existence of a certain degree of hypotension which seems to be the rule after 80 years of age, even if the systemic arterial pressure is normal. This condition should be compared with the fact that in the aged the ocular tension is usually low. Wessely considered such a low ocular tension to be proof of sclerosis of the peripheral vessels.

In the healthy adult the diastolic retinal pressure is about 35 mm. of mercury and the systolic pressure from 70 to 90 mm. All writers have not agreed with these figures. The reasons must be sought in the use of a technic lacking in precision at a time when the method was little known as well as in the use of varying criteria, some taking the figure corresponding to the first pulsation and others that for the first transitional arterial collapse. This is why, instead of looking for the first tremor of the artery, which is difficult to perceive, it is better to compress the eyeball until the pulsation is distinct without collapse of the vessel. This frank pulsation is to be correlated with the average pressure in the brachial artery; the figure is about 40 mm. of mercury.

However, I must further insist that only similar things are to be compared and that only a relative value is to be attributed to the ratios. It cannot be otherwise because the comparison curve that I prepared with Bailliart, which serves to calibrate in millimeters of mercury the retinal pressure readings of the instrument, was derived from experimental data on animals and not on man.

It is important that compression of the eyeball be made fairly rapidly. A slow compression causes a reflex increase in the blood pressure.

In a normal subject the retinal arterial pressure is constant. But since it varies with the cerebral pressure, emotion or fear causes an increase in the pressure which may last half an hour (Dubar). This increase is accompanied by a greater frequency of the pulse, and affects the systolic pressure much more than the diastolic. The average pressure does not vary. In man, injections of epinephrine increase at the

same time both the general and retinal pressure without modifying the caliber of the central artery. But if acetylcholine is injected at the same time, the artery dilates (Villaret, Schiff and Justin-Besançon).

Tonoscopy of the Vein.—Indications derived from tonoscopy of the central vein have not the same value as those supplied by tonoscopy of the artery. The venous pressure is not so constant and, without going outside of physiologic limits, it may be slightly superior or inferior to the ocular tension. Very frequently venous pressure and ophthalmotonus balance each other and then the vein shows a spontaneous pulse. The character of this pulse is very peculiar; the vein seems to fill and empty passively, collapsing slowly without any appearance of active contraction and filling still more slowly. The same contrast is present in the cardiac cycle; the artery dilates at the moment of the systole, and it is at this moment that the vein collapses. An instant later when the diastolic contraction of the artery takes place the vein begins to fill. This spontaneous venous pulse, which must not be confused with certain oscillations transmitted to the vein by the artery because of its proximity, exists in one case out of three. When it does not exist its appearance can sometimes be provoked by pressing slightly on the eyeball, but at other times no amount of compression can cause its appearance. Therefore three eventualities of equal frequence are met with: spontaneous pulse, provoked pulse and absence of venous pulse.

Duke-Elder has confirmed by delicate experiments Bailliart's opinion that the pressure in the intra-ocular veins is scarcely higher than the ophthalmotonus and that the eye falls into line with the rest of the body where it is known that the venous pressure is slightly higher than the tissue pressure. Therefore the three eventualities just set forth may be explained as follows:

(*a*) Spontaneous Venous Pulse: The venous pressure and ophthalmotonus balance each other. The venous pressure exceeds the ophthalmotonus during the diastole and the ophthalmotonus exceeds the venous pressure during the systole.

(*b*) Induced Venous Pulse: The venous pressure is higher than the ophthalmotonus. The application of a small amount of pressure on the eyeball raises the ophthalmotonus; equilibrium is produced and the pulse appears.

(*c*) Absence of Pulse: The venous pressure is slightly lower than the ocular tension. By increasing the ophthalmotonus one increases the difference between the two pressures, and if one continues to press on the eyeball the arterial pressure is increased until the pressure level of the carotid artery has been reached.

It requires a pressure of from 70 to 80 mm. of mercury for the veins in those circumstances to be diminished in volume when the diastolic

arterial pressure is greatly exceeded (Duke-Elder). This explains why the intra-ocular venous pressure may be slightly less than the ophthalmotonus without the observer being able to distinguish any change in caliber. As to the blood flow, it is assured by the vis a tergo issuing from the capillaries.

Thus, from the tonoscopic point of view, examination of the central vein gives information only if there is spontaneous or provoked pulse.

The arterial pressure is only slightly and indirectly influenced by posture. The venous pressure, however, is influenced by the position of the head; it increases slightly in the recumbent posture, and still more when the head is very low. The normal respiratory movements have little influence, but apnea with effort provokes a notable increase (Dubar and Lamache). Cold has a very variable action.

Although the relations between the venous and intracranial pressures are looser than those between the intracranial and the retinal arterial pressures, nevertheless compression of the jugular veins (Queckenstedt-Stookey) induces an increase in the pressure of the central vein. The rise is slow and reaches its maximum only after ten or twenty seconds.

TONOSCOPY AND INTRACRANIAL HYPERTENSION

Clinical experience having shown a close relation to exist between the retinal arterial pressure and the cerebral circulation, it is at once apparent that tonoscopy is especially important in intracranial diseases. It was early noted that hypertension of the spinal fluid was accompanied by a dissociation between the general arterial pressure and the pressure in the central artery. Until the time of these observations, unless a lumbar puncture was made, intracranial hypertension could only be discovered by the appearance of choked disk. Thus, by means of tonoscopy the physician may obtain definite information on which to base a decision to make a lumbar puncture and a study of the spinal fluid. Numerous cases of serous meningitis in which a choked disk never developed have been discovered in this way (Bailliart, Magitot, Coppez, Kalt, Claude, Lamache and Dubar, Winther).

The characteristic finding in serous meningitis is an increase in spinal fluid, but contrary to the condition in other forms of meningitis this fluid is chemically normal. The condition may have a traumatic or non-traumatic origin, and has the aspect of a true cerebral glaucoma.

Tonoscopy has made it possible to observe a certain number of these intracranial hypertensions after shock. Coppez reported a case diagnosed before the appearance of choked disk; Bailliart and Merigot examined a patient with a fracture of the skull who had intracranial hypertension and an increase in retinal pressure for a week. Claude, Lamache, Dubar, Kalt, Fritz, Arnould, Magitot and Winther have published similar observations.

However, if in practice tonoscopy has made the ophthalmologist better acquainted with a condition that was considered as exceptional, Bailliart's method has also shown itself useful for detecting intracranial hypertensions and especially those resulting from the presence of tumors. Claude, Lamache and Dubar in 1928 reported twenty-six cases, and since then numerous writers have contributed to the subject (Dubar, Kalt, Magitot, Gallois, Gaudissart, Winther).

The most diverse cases may present themselves, and here are a few examples:

I observed a woman, aged 39, suffering from epileptic fits. As she had been operated on some time previously for a tumor of the breast, it was thought that there might be metastases. I was soon convinced, however, that it was a case of syphilitic meningitis. The increase in the retinal arterial pressure was confirmed by three lumbar punctures. The Wassermann reaction being negative in the third, she was treated and cured in six months.

A second case concerns a patient who complained of giddiness and headache. Apart from a paralysis of the third nerve on one side, the ocular examination had given negative results, but the retinal arterial pressure permitted a diagnosis of intracranial hypertension. A lumbar puncture was made and the case proved to be one of tuberculous meningitis. In this malady choked disk is rare but, according to the tonoscopic examinations that I have carried out on several patients under treatment in the medical wards; intracranial hypertension appears to be the rule.

A third case was that of a woman, aged 46, who had been operated on for cancer of the breast eighteen months before and who showed clinical symptoms of a tumor of the cerebellum. The eyes were normal except for a slight anisocoria and a high retinal arterial pressure. Lumbar puncture revealed a spinal hypertension. During two days the arterial pressure on the disk remained low; then it increased again The patient died ten days later, and postmortem examination revealed the presence of four metastatic tumors, three in the cerebellum and one in the frontal lobe. Winther and Bauvens have brought forward similar observations.

A fourth case was that of a former soldier who had had a severe concussion of the brain. Since 1919 he had been suffering from frequent epileptic fits. In 1927 the Wassermann reaction was negative several times and the eye was normal, but as tonoscopy had shown an increase in the retinal arterial pressure a lumbar puncture was made. The pressure was normal by the manometer, and compression of the jugular veins was followed by an increase in the pressure. On analysis, the fluid was also normal. However, as the patient still suffered, the third ventricle was punctured and the fluid at first spurted forth, and then flowed drop

by drop. This case is interesting because it shows that there may be hypertension in the ventricles without lumbar hypertension. Kalt, in 1927, also reported the history of a patient in whom, outside of the subjective syndrome, the retinal hypertension was, for several months, the only sign of intracranial hypertension. Lumbar puncture had given negative results. An operation showed the existence of a circumscribed arachnoiditis.

When Queckenstedt's test is negative the value of tonoscopy is increased. Claude, Lamache and Dubar reported two cases in which the retinal pressure was high, and lumbar puncture as well as compression of the jugular veins gave negative results. In one case puncture of the third ventricle was made and the pressure of the fluid by Claude's manometer was 38 mm. of water. The other patient had syphilitic meningitis and was treated and cured in three months. Similar observations are found in Winther's paper.

These facts show that compression of the jugular veins provokes normally not only an increase in the intracranial pressure but, at the same time, an increase of the pressure in the central artery (Claude, Lamache, Dubar, Berens). This modification of the pressure takes place after a certain delay, and the explanation seems to be that the ophthalmic artery before penetrating the orbit is surrounded by fluid. The fluid, the pressure of which may be increased by Queckenstedt's technic, exercises a certain degree of compression on all the arteries at the base of the brain. It is probable that, as was shown by Cushing, the vasomotor centers in order to overcome this increased resistance automatically induce an increase in the blood pressure.

The same effect is obtained when the brain is compressed through the opening of a former trephining (Bailliart and Hartmann, Lamache and Dubar). I myself have been able to verify the truth of this observation, for, by pressure on the temporal region of a man who had been wounded and had a wide bony breach, an increase in the retinal arterial pressure was induced. Inversely, in cases of intracranial hypertension when hypertonic salt solution is injected in accordance with Weed and MacKibben's technic, a moderate diminution of the intraspinal pressure and of the retinal pressure is noted (Claude, Lamache, Dubar, Kalt, Gaudissart).

It would seem, moreover, that manifestations of activity of the cerebral vasomotor centers may take place at the same time as, or independently of, a modification in the pressure of the spinal fluid. Claude, Lamache and Dubar, who have examined by the tonoscopic method more than 250 patients in their clinic for mental diseases, have observed that, contrary to what exists in normal subjects, a certain number of patients show circulatory disturbances manifested by a change of pressure in the retinal artery. In epileptic patients such hypertensions are indepen-

dent of emotion and changes in the general pressure and manifest themselves outside of the fits (Dubar, Winther). On the contrary, in certain psychoneuroses tonoscopy enables one to recognize a retinal hypotension with spasm.

Hypotension has, indeed, drawn the attention of these writers, for in certain patients a low pressure of the spinal fluid is accompanied by giddiness, asthenia, headache and low retinal pressure. More study and observations on this point would be of great interest.

The pressures in the central artery and in the spinal fluid show, therefore, a striking parallelism, a fact that has been confirmed recently by the observations of Berens, Smith and Cornwall. But it must be borne in mind that tonoscopy only gives an indication; it cannot enable one to estimate in figures the value of the intracranial pressure, and I have pointed out that a very high intracranial pressure might be accompanied by only a moderate rise of the retinal pressure and vice versa. Moreover, normally after lumbar or cisternal puncture tonoscopy shows that the pressure on the disk does not fall immediately. After falling, sometimes one or two hours are required for the pressure to regain its normal value. Occasionally it happens that after having fallen it rises, falls and rises again. These facts are interesting to note because they must correspond to modifications in the intraspinal pressure similar to those observed in the ocular tension. This phenomenon also takes place in the eye after paracentesis of the anterior chamber; it is a reactive hypertension. It has been described by Guillain, and I drew attention to its meaning in the *Revue neurologique* (1927). Recently it has been described again by Claude, Lamache and Dubar. Sometimes when the fluid has been removed too rapidly cephalgia and vomiting may occur, symptoms that express the disturbance in the cerebral circulation.

Tonoscopy, because it is able to reveal intracranial circulatory disturbances, is an important method for the study of the much discussed cerebral vasomotor phenomena. Indeed, it must be remembered that physiologists have, up to the present, denied the existence of an innervation of the cerebral arteries, whereas neurologists assert the contrary. But by careful and elaborate studies, Stohr discovered nerve fibers in the walls of the arterioles and capillaries of the brain, and the experiments of Stanley Cobb, La Fontaine Verwey and Tinel have confirmed the opinion of the neurologists. Although there is not complete agreement on the question, Tinel has recently brought forward evidence to show that the regulation of the cerebral circulation is assured by the autonomic system. Section of the cervical sympathetic enables epinephrine, normally without action, to cause an intense vasoconstriction.

Together with Bailliart, I studied in animals the action of the sympathetic on the retinal artery. We were able to see those modifications of its caliber which Lériche, Dubar and Lamache have noted in man.

These observations should be compared with those of Claude, Lamache and Dubar who occasionally found in migraine a diminution in the pressure of the central artery.

I have been discussing arterial tonoscopy; I must now mention venous tonoscopy. On account of the weak pressure in the veins, its study is difficult and more of theoretical than practical value. Baurmann had thought he could deduce, from the absence of venous pulse, the presence of intracranial hypertension, but this is incorrect since a spontaneous pulse does not exist in two thirds of normal persons.

The retinal venous pressure reflects, however, fairly faithfully variations in intracranial and intraspinal pressure. One may prove this easily by Queckenstedt's test. Compression of the neck raises the intracranial pressure by from 10 to 30 mm. of water and the pressure in the central vein by 10 mm. The ligation of one jugular vein has the same effect (Claude, Lamache, Dubar). Yet in certain psychoneuroses there is no relation between the retinal and venous pressure and the pressure of the spinal fluid (Claude, Dubar, Lamache). The former may be high and the latter low. In other patients tonoscopy shows modifications when the patient passes from the sitting to the recumbent posture. Bailliart has observed an increase in the venous pressure of the pulsatile type in two cases of migraine.

One has now to study the information that tonoscopy gives in cases of choked disk. Choked disk being a sign of intracranial hypertension, one should expect to find a high retinal pressure. Yet clinical studies show that it is accompanied by a chain of peculiar pressure relationships. In a first stage the intraspinal tension rises progressively and the retinal arterial pressure, without having an exactly parallel value, follows this rising curve. During several weeks there is a relative stabilization at the high figure, and then in the third stage, almost at the same time as the choked disk appears, the diastolic retinal pressure suddenly collapses without any concomitant variation in the intraspinal pressure (Claude, Lamache, Dubar). Thus, as has been noted by most writers, in choked disk the retinal arterial pressure is hardly above normal (Bailliart, Dubar, Berens, Winther and others) and is sometimes even inferior to the normal figure (Deyl). As to the venous pressure it is scarcely modified, a fact that does not agree with the theory expressed by Deyl and Dupuy-Dutemps who attributed the edema of the disk to a compression of the central vein by the spinal fluid at its entrance into the optic nerve.

In reality, one should not lose sight of the fact that a choked disk is the manifestation of an edema. I rather believe in the theory that postulates an extension to the chiasm and the optic nerves of an edema coming down from the region of the tuber (Parinaud, Ulrich, Rochon-Duvigneaud, Sourdille, Kampherstein, Uhthoff, Bollack). The edema itself is due to an increase in the tissue fluid, a fluid similar to the spinal

fluid and the aqueous humor, which is dialyzed by the capillaries. The edema and often the small hemorrhages that are met with around the disk are therefore attributable to an increase in the permeability of the walls of the capillaries. This modification of the normal physiologic conditions is accompanied by a vasodilatation, the effect of which is to lower the arterial pressure. The same phenomenon exists in edema of the lung. The true cause of this functional asystole should be sought in a disturbance of the local vasomotor innervation, a repercussion of the disturbance of the vasomotor innervation of the cerebral vessels.

TONOSCOPY IN DISEASES OF THE EYE

The retinal circulation, while being a reflection of the cerebral circulation, may likewise be disturbed by purely local conditions.

Systemic Hypertension (Malignant Hypertension, Nephritis).—In cases of systemic hypertension the edema and the exudates that are met with in the retina are more extensive and more abundant than in choked disk of intracranial origin. This is, indeed, about the sole difference existing between the two, and numerous writers have long since called attention to the impossible position that one would be in, if in the absence of accessory signs, one were obliged to base the diagnosis solely on the ophthalmoscopic examination. Evidently it is merely a question of degree.

Retinitis of the albuminuric type is observed in Bright's disease and in diabetes. It is also found in patients who have neither hyperglycemia nor azotemia, but who are suffering from a very high general blood pressure. For a long time it was thought that this systemic hypertension was due to kidney disease and that the toxemia resulting from it produced this retinitis. But nowadays pathologists admit the existence of a disease peculiar to the blood vessels, characterized by a high blood pressure (Wagener, Dumas), which in its terminal stages is accompanied by renal and ocular lesions. This is the reason why the appearance of edema and of exudates makes the diagnosis so serious.

On the other hand, it has been observed that in patients suffering from albuminuric retinitis and high blood pressure there is a hypertension of the spinal fluid. This fact originated the thought that the edema of the retina might, as in cases of cerebral tumor, be produced by the intracranial hypertension. Accordingly, Cushing and Bordley, von Hippel and Guillain have proposed lumbar puncture and even decompressive craniectomy as a prophylactic ocular treatment.

As a matter of fact, the systemic hypertension of the simple form, namely, that without ocular lesions, is sometimes accompanied by intracranial hypertension which manifests itself on the disk by an increase in the retinal pressure (Bailliart, Magniel and Saragea). These patients, however, frequently show an inversion of the tonoscopic formula. No

special picture for the fundus of the eye exists, although certain patients sometimes complain of small bright or black subjective spots or even of visual eclipses which are to be regarded as signs of a spasmodic state. In some there appear exudates and slight hemorrhages preceding the nephritic signs, whereas in others, according to the classic picture, the nephritic signs are associated with ocular lesions; but then, as in choked disk of intracranial origin, one notices a rather low retinal arterial pressure.

Three categories of ocular findings associated with systemic hypertension may therefore be distinguished (Abrami, Gallois, Stehelin):

1. A systemic hypertension without an increase in the retinal arterial pressure. In these no retinal lesions are found. 2. A systemic hypertension with an increase in the retinal pressure. In these also there is no retinal lesion, but there is a low ocular tension, a sign of a peripheral spasmodic state. 3. A systemic hypertension, a moderate increase in the retinal pressure and ocular lesions. This state may precede or be followed by nephritic symptoms.

In the second and third categories certain writers think that the increase in the retinal pressure is due to a hypertension of the spinal fluid. Others, for instance, Lamache and Dubar, have attributed the increase to local lesions of the vessels. I agree with Bailliart in believing in the importance of the vascular lesions and I think that when they become generalized in the brain they induce the increase in the spinal fluid. In the eye these lesions involve at first principally the capillaries. They cause exudates, and it must be remarked that they are not solely localized in the retina; they are likewise found disseminated in the choroid (Rochon-Duvigneaud, Wagener).

Wagener has described four anatomopathologic phases in the hypertensive syndrome. In the first stage one notes retinal and subretinal edema, hyperemia and edema of the disk and, around the disk, a few hemorrhages which are usually superficial, and small exudates.

In the second stage the edema extends over the surface and invades the macular region. The exudates are more abundant and of a cotton-wool aspect. Hemorrhages are found in the deeper layers of the retina.

In the third stage the lesions spread over the entire retina and invade the choroid. Brownish-black spots of pigment and atrophy of the choriocapillaries are to be seen. The optic nerve begins to atrophy. A few stellar exudates exist in the macular region.

In the fourth stage the optic atrophy is complete; there are few hemorrhages and exudates; the retina atrophies. There are disseminated spots of pigment, sclerosis of the arteries and a thickening of the veins. Wagener stated that the thickening of the arterial walls is not due to a hyperplasia of the intima but is induced by fibrosis around the vessel combined with a slight medial hypertrophy. These lesions are more

marked in the choroid than in the retina. They end in a lipoid degeneration of the vessel walls. In three patients they were observed also in the brain and in the kidneys.

Sclerosis of the Vessels.—In case of sclerosis of the retinal vessels (Verwey) tonoscopy can supply two kinds of information: the degree of the hydrodynamic pressure and the state of rigidity of the artery.

Senile modifications are characterized by a diminution in the strength of the provoked pulse. It is the normal state of a person of 80, but the change becomes pathologic in younger patients and is often accompanied by objective modifications in the caliber of the vessels.

The state of rigidity of the artery can be estimated by its resistance to compression, using a technic developed by Fritz at the eye clinic of Professor Coppez in Brussels as a result of his plethysmo-oscillographic researches. Bailliart's dynamometer is applied to the eyeball, and the reading at the first beat is noted. Pressure on the eyeball is continued until the artery, beating more and more strongly, empties and fills rhythmically. The new reading is noted. The difference between the two figures measures the resistance of the artery to compression.

This resistance to compression is influenced by different factors. Thus a high blood pressure, an increase in the viscosity of the blood or an enlargement of the caliber of the vessel intervenes as a secondary element, whereas the rigidity of the walls of the vessel plays the essential part. Fritz found that in normal man the resistance to compression was always less than 10 Gm. In the child one may find 5 Gm. only, while in a man aged 77 Fritz found 8 Gm. When the retinal pressure is increased without there being any lesion of the artery, as in the case of concussion of the brain with intracranial hypertension, the resistance to compression does not exceed 10 Gm. On the contrary, in patients affected with malignant hypertension, not only does tonoscopy reveal an increase in the retinal pressure, but the resistance to compression of the artery exceeds 10 Gm., and may reach 20 Gm., indicating an advanced state of sclerosis.

Fritz was able to measure the resistance of the artery in different diseases: in thrombosis of the central vein, in syphilitic arteries and in multiple sclerosis with retrobulbar neuritis. This new method demands a certain amount of training on the part of the ophthalmologist, but the results described by this writer appear to me to be of quite a little interest.

Even when the systemic pressure is normal the obliteration of a retinal branch is usually accompanied by an increased pressure in the arterial trunk (Stasnicki). However, the lesion of the ocular vessels ends finally in a diminution of the arterial pressure. Bailliart has drawn attention to this in the evolution of optic atrophies in acromegaly. I have noted a marked hypotension coinciding with a low systemic pressure

in a case of posthemorrhagic blindness. However, it was not possible to say whether or not there existed any lesions of the retinal vessels.

Glaucoma; Detachment of the Retina.—The tonoscopic modifications in glaucoma are very interesting and concern the arterial and venous pressures. As Bailliart stated in his book on the retinal circulation and as we have both observed in studying the circulatory conditions in glaucoma, the characteristic of this malady is a moderate rise in the diastolic arterial pressure and a great increase in the venous pressure. These two changes call for a few comments.

(*a*) Arterial Pressure: In the majority of cases the arterial diastolic pressure has a tendency to harmonize with the ophthalmotonus. It is influenced, in a certain measure, by the general pressure and especially by the cerebral pressure. The proof of this is the increase in the ophthalmotonus in glaucomatous patients (contrary to what takes place in the normal individual) when they change from the sitting to the recumbent posture (Thibert). Similarly, the retinal arterial pressure follows, as a rule, the modifications of the ophthalmotonus induced by therapeutic means. When the ocular tension exceeds the retinal diastolic pressure one sees the spontaneous pulse appear, a phenomenon that is found more frequently in acute than in chronic glaucoma. As far as the systolic arterial pressure is concerned, and although it is difficult to estimate on account of the turbidity of the vitreous which is exaggerated under compression, one may likewise consider that it follows the ophthalmotonus which it always exceeds.

To sum up, whether the general blood pressure be normal or high, in the glaucomatous eye the blood pressure in the central artery follows the ocular tension. The diastolic pressure, but not the systolic pressure, may be lower than the ophthalmotonus. The differential pressure between the systolic and the diastolic is feeble.

(*b*) Venous Pressure: The venous pressure may, in glaucoma as in the normal state, be higher, equal or slightly lower than the ocular tension. When it is in equilibrium with the ophthalmotonus there is a spontaneous venous pulse (13 per cent of cases, Bailliart). But since in glaucoma the ocular tension is generally high, one may therefore conclude that the venous pressure is likewise high. Bailliart also pointed out that a venous pulse appears with weak ocular tension and high arterial pressure. On the other hand, an arterial pulse always appears with high ocular tension. The local blood pressure is therefore always high in glaucoma.

Moreover, in order to form an estimate of the circulatory disturbance occurring in a glaucomatous eye, it suffices to take the tension of an enucleated globe. In a normal eye the tension during life is 25 mm. of mercury. After enucleation the tension is still nearly 10 mm. of

mercury. The difference between 10 and 25 mm. indicates the importance of the circulatory factor. In a glaucomatous eye before operation the tension was 60 mm. of mercury. After enucleation the tension was 20 mm. of mercury. The difference between 20 and 60 mm. of mercury shows what a great part the local blood pressure plays in this disease.

On the other hand, it may be recalled that sclerosis of the vessels, especially of the veins, has long since been described as an anatomic observation. But as I have set forth on many occasions, for instance, in the *Annales d'oculistique* (1929), glaucoma must be considered as essentially due to a disturbance of the capillary permeability. This disturbance is accompanied by an increase in the blood pressure in the capillary network and by an increase in the permeability of the walls of these vessels, vessels which dialyze the aqueous humor. As the aqueous humor is merely a tissue fluid, the quantity that transudes accumulates in the tissues, uvea, retina and vitreous, and this accumulation constitutes the edema. It is the increase in the blood pressure in the capillaries and the effusion of fluid into the tissues that cause the ophthalmotonus to increase. This condition reacts on the arteries which build up pressure to overcome the obstacle, and as the pressure rises that of the capillary network increases still more. Thus the diastolic pressure can be exceeded by the ocular tension. The ophthalmotonus follows the systolic pressure, and the systolic pressure rises automatically until the pressure level of the carotid has been reached.

At other times the increase in blood pressure resulting from the capillary resistance may lead to a certain degree of local asystolia. Then the diastolic pressure goes down until it is balanced by the ocular tension and a spontaneous pulse appears. Edema of the vitreous prevents one from seeing this pulse, which must exist more frequently than has been described.

Operative decompression causes a fall in ocular tension and arterial pressure, although the blood pressure remains high in the capillary network. The entoptic studies of Dieter give proof of this condition. Ocular hypertension is, indeed, merely one of the symptoms of glaucoma. Glaucoma can progress toward blindness without the tension ever exceeding 35 mm. of mercury, and all ophthalmologists know that an operation that lowers the tension is often powerless to arrest the optic atrophy. The reason is that the operation, in spite of the changes which it initiates, is sometimes totally incapable of reestablishing a normal circulation in the diseased capillary network. Tonoscopy reveals in such cases a low retinal pressure.

In detachment of the retina, since the ocular tension is often low, one should likewise expect to find a low blood pressure, but, on the contrary, tonoscopy reveals a fairly high retinal pressure (Tille).

Retrobulbar Neuritis.—Both Winther and Colrat have reported cases of retrobulbar neuritis with retinal hypertension.

As may be seen from this article, tonoscopy has, in a very few years, enabled one to acquire interesting information, concerning not only the ocular, but also the intracranial circulation. For the therapeutist, it therefore completes in a useful manner the study of the retinal vessels. Its principal interest, outside of ophthalmology, is to supply the neurologist with information as to the state of the intracranial pressure. Although it is a young science and capable of being improved, the method is worthy of being examined by all ophthalmologists, and I take it that in a few years it will become as current as the act of determining systemic pressure with the sphygmomanometer or ocular tension with the tonometer.

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ISO-ELECTRIC POINT OF LENS PROTEIN *

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From the report of Scalinci,[1] it appears that Gullotta was the first to make an attempt to determine the iso-electric point of lens protein. Gullotta's method in brief was as follows: Solutions of different hydrogen ion concentrations were prepared with acetic acid and sodium acetate. Ox lenses were kept in these solutions for a certain period of time, and the increase in weight was ascertained at the end of the experiment. The minimum absorption of water was found to occur at p_H 4, and this was regarded as the iso-electric point of the lens protein. Scalinci, using Gullotta's method, made a separate determination of the iso-electric point of the cortex and nucleus of the lens. He reported the following p_H values: cortex, from 3.5 to 4; nucleus, 4.5. This gives an average p_H value of 4 for the whole lens protein and agrees with the results obtained by Gullotta. Woods,[2] on the other hand, worked on alpha and beta crystallines and found the respective iso-electric points to be 4.8 and 6 on the p_H scale. The range of maximum precipitation for the whole protein was found by Woods and Burky[3] to lie between 4.8 and 5.2 on the p_H scale. According to the work of Gonçalves,[4] the protein of the whole lens has two iso-electric points. Minimum absorption of water by the lens protein was observed at p_H 4.6 and p_H 8.5. Correspondingly, Gonçalves found also that the buffer effect of the protein was at its minimum at 4.6 and 8.3 on the p_H scale.

Whereas there is excellent agreement between the results of Gullotta and Scalinci, on the one hand, and fair agreement between the results of Woods and Gonçalves, on the other hand, the observations for the two

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groups differ widely. The difference evidently is due to the employment of different methods.

While working on the turgescence and deturgescence of the vitreous protein, we [5] realized that the hydrogen and hydroxyl ions of the outside solution penetrate the vitreous gel with great difficulty. This must be even more true with respect to the crystalline lens on account of its greater density. Unless the lens is broken into minute particles and the protein allowed to become completely saturated with a reagent solution of different hydrogen ion concentration from that of the lens, the reaction of the surrounding fluid is different from that within the lens.

It appears from the available literature that, at least in some cases, the importance of this point may have been neglected. In the present work, therefore, special care was taken to have the lens protein completely saturated with the reagent solution.

METHOD

The lenses of cattle from 1 to 2 years old were used in all experiments. The eyes were enucleated immediately after the death of the animals and kept at a temperature slightly above the freezing point until removal and preparation of the lenses could be accomplished. Usually three hours elapsed between the death of the animals and the experiment. After the lenses, or parts of them (nucleus or cortex), were rubbed in a mortar to a more or less homogeneous paste, sufficient distilled water was added to give an easily flowing emulsion. This emulsion was strained through four layers of ordinary hospital gauze; thus the larger particles were collected on the gauze. The gauze was then wrapped around this residue and the whole returned to the mortar and macerated in small amounts of distilled water and the process repeated until practically the entire material was washed through the gauze. The resultant emulsion was added each time to the first collection and the whole finally made up to such a volume that 20 cc. contained from one-half to two whole lenses. Exactly 20 cc. of the emulsion was pipetted into each of several previously prepared collodion tubes. (The technic is described in detail in a previous article.[4]) Each tube was then suspended in a 500 cc. Erlenmeyer flask containing 475 cc. of a hydrochloric acid solution of known hydrogen ion concentration. Usually eight hydrochloric acid solutions of different hydrogen ion concentrations were employed, ranging from p_H 2.5 to p_H 7 (the hydrogen ion concentration of distilled water). In some experiments solutions of sodium hydroxide of different hydroxyl ion concentrations up to p_H of about 9 were employed. The tubes with their contents were allowed to remain in solutions for about twelve hours. At the end of this period there were always certain tubes in which the lens protein had more or less completely coagulated. In other tubes the lens protein was found to have gone more or less completely into solution. The contents were then transferred into 50 cc. graduated tubes, all of which were centrifugated for the same period of time at the same speed. The supernatant fluid was used in the determination of the hydrogen ion concentration. This was done electrometrically with a saturated calomel cell and the

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hydrogen electrode. Small quantities of amyl alcohol were allowed to slide down the outer jacket of the electrode in order to prevent frothing when the hydrogen gas was passed through the solution.

The p_H associated with the largest amount of precipitate was assumed to be the iso-electric point of the lens protein. Such an assumption is justified on the following grounds: 1. Hitchcock,[6] who determined the iso-electric point of gelatin, showed that maximum opacity (or precipitation) coincided with minimum osmotic pressure, the former being at 5.05, the latter at 5.02, on the p_H scale. 2. Lothrop and Paine[7] determined by cataphoresis that the colloids of honey had no electric charge (or had the least ionization) at p_H 4.3. Coincidentally, they observed also that at this hydrogen ion concentration the turbidity (precipitation) was at its maximum. 3. Van Nostrand's Chemical Annual[8] defines the iso-electric point of a protein as follows: "For substances that are amphoteric in nature, and have a high degree of hydration, the I. E. P. represents the H^+ concentration at which

(*a*) the tendency to precipitate out is at a maximum,
while (*b*) the degree of ionization
(*c*) the degree of swelling
(*d*) the degree of hydration
(*e*) the viscosity
(*f*) the osmotic pressure

are at a minimum."

In order to check the accuracy of the results obtained with collodion tubes, the procedure was slightly modified in a few experiments. Exactly 5 cc. of lens emulsions, containing from one-half to one entire lens, was pipetted into each of several 50 cc. centrifuge tubes. Into each tube was delivered 40 cc. of a reagent solution differing in reaction from every other solution employed. The tubes were allowed to stand over night and then were centrifugated, following which the p_H of the supernatant fluid was determined and the volume of the precipitate measured, as described in the first procedure. The results of both methods were practically the same.

RESULTS

In tables 1 to 6 inclusive are listed the data obtained in experiments in which the precipitation was carried out in collodion tubes before the contents were transferred to 50 cc. centrifuge tubes. In the remainder of the experiments (tables 7 to 10, inclusive) graduated 50 cc. centrifuge tubes were employed throughout the experiment; that is, both precipitation and centrifugation were carried out in the centrifuge tubes. The data shown in the first eight tables were obtained on the whole lens; those in tables 9 and 10 were obtained on the cortex and nucleus, respectively. No attempt was made to find in each experiment the exact value of the iso-electric point of the lens protein, since such an attempt would

6. Hitchcock, D. I.: New Determination of the Iso-Electric Point and Combining Capacity of a Purified Gelatin, J. Gen. Physiol. **12**:495, 1929.

7. Lothrop, R. E., and Paine, H. S.: Some Properties of Honey Colloids and the Removal of Colloids from Honey with Bentonite, J. Indust. & Engin. Chem. **23**:328, 1931.

8. Van Nostrand's Chemical Annual, ed. 6, New York, D. Van Nostrand Company, 1926, p. 484.

require a very large number of reagent solutions differing in hydrogen ion concentration from each other by only a few small units. The p_H values printed in heavy type are assumed to be the nearest to the iso-electric point. The average of such results based on several experiments gives an accurate value for the iso-electric point. In cases in which two tubes (in the region of the iso-electric point) in the same experiment had nearly equal amounts of precipitate, both p_H values were used in the

TABLE 1.—*Each Collodion Tube Contained Approximately 1.5 Lens (Whole Lens)*

Number of tubes	1	2	3	4	5	6	7	8	9
Precipitate, cc.	0.0	0.25	4.5	**8.0**	3.5	2.5	1.25	0.8	0.8
p_H	2.35	3.28	4.63	**4.87**	6.35	6.47	6.96	7.27	7.50

TABLE 2.—*Each Collodion Tube Contained Approximately 1.5 Lens (Whole Lens)*

Number of tubes	1	2	3	4	5	6	7	8
Precipitate, cc.	0.1	2.0	5.0	**7.5**	3.5	0.8	0.3	0.1
p_H	3.36	4.33	4.75	**5.48**	6.25	7.25	8.23	9.21

TABLE 3.—*Each Collodion Tube Contained Approximately 2 Lenses (Whole Lens)*

Number of tubes	1	2	3	4	5	6	7	8	9
Precipitate, cc.	0.2	7.5	**11.0**	5.0	3.5	2.5	0.5	Trace	Trace
p_H	3.55	4.85	**5.39**	6.02	6.37	6.71	7.36	7.99	9.53

TABLE 4.—*Each Collodion Tube Contained More Than 2 Lenses (Whole Lens)*

Number of tubes	1	2	3	4	5	6	7	8	9	10
Precipitate, cc.	0.1	9.5	**20.0**	11.0	3.5	0.25	0.25	0.25	0	0
p_H	3.60	4.56	**4.88**	5.86	6.32	6.76	6.83	7.01	9.51	9.70

TABLE 5.—*Each Collodion Tube Contained Approximately 1 Lens (Whole Lens)*

Number of tubes	1	2	3	4	5	6	7	8	9	10
Precipitate, cc.	3.0	7.5	**11.0**	7.25	5.0	3.0	3.0	2.5	1.0	1.0
p_H	4.05	4.50	**5.40**	5.75	6.25	6.70	6.80	7.10	9.50	9.90

computation of the average. The iso-electric point of lens protein, obtained by precipitation in collodion tubes, was found to be at an average p_H 5.11 (tables 1 to 6, inclusive). In similar experiments carried out by precipitation in centrifuge tubes the iso-electric point was at an average p_H 5.25 (tables 7 and 8). The results obtained by the two procedures are in close agreement. The average of all eight experiments (tables 1 to 8, inclusive) is 5.16. One experiment was carried out on the cortex, another on the nucleus. The iso-electric points were found to be p_H 5.10 and p_H 5.44, respectively. This is in agreement with the

observations of Scalinci,[1] who found a similar difference in the iso-electric points of these two parts of the lens protein. On either side of the iso-electric point the lens protein passed more or less completely into solution, quite rapidly when the hydrogen ion concentration was increased, more slowly when the hydrogen ion concentration was decreased or the hydroxyl ion concentration was increased to about p_H 9. At p_H of about 8.5 only a small fraction of the lens protein came

TABLE 6.—*Each Collodion Tube Contained Approximately 1 Lens (Whole Lens)*

Number of tubes	1	2	3	4	5	6	7	8
Precipitate, cc.	0.2	2.0	9.0	8.5	6.5	1.5	1.5	0.6
p_H	3.70	4.10	4.65	5.10	5.70	6.50	6.40	6.70

TABLE 7.—*Each Centrifuge Tube Contained Approximately 1 Lens (Whole Lens)*

Number of tubes	1	2	3	4	5	6	7	8
Precipitate, cc.	0	0.2	5.5	6.0	4.0	0.75	0.5	0.4
p_H	2.30	3.10	5.05	5.40	6.00	6.60	6.90	7.10

TABLE 8.—*Each Centrifuge Tube Contained Approximately 1 Lens (Whole Lens)*

Number of tubes	1	2	3	4	5	6	7	8
Precipitate, cc.	0.1	0.2	0.5	7.5	7.0	0.75	0.5	0.4
p_H	2.10	2.90	4.00	5.00	5.55	6.40	6.70	7.10

TABLE 9.—*Each Centrifuge Tube Contained Approximately 0.5 Lens (Cortex)*

Number of tubes	1	2	3	4	5	6	7
Precipitate, cc.	0.1	0.2	2.0	2.5	1.75	1.1	1.0
p_H	2.1	2.9	4.9	5.1	5.8	6.6	7.2

TABLE 10.—*Each Centrifuge Tube Contained Approximately 1 Lens (Nucleus)*

Number of tubes	1	2	3	4	5	6
Precipitate, cc.	0.1	3.5	7.0	6.5	5.1	0.3
p_H	3.8	4.6	5.27	5.60	6.3	6.9

down as a precipitate. This observation is entirely at variance with that of Gonçalves,[4] who reported having observed a second iso-electric point at p_H 8.5.

SUMMARY

This paper contains a description of a simple and accurate method by which the iso-electric point of the lens protein may be determined. The iso-electric point of the protein in the whole lens of cattle was found to be at p_H 5.16. We could not corroborate Gonçalves'[4] report of a second iso-electric point at about p_H 8.5. In agreement with the results

obtained by Scalinci,[1] the iso-electric point of the nucleus is at a slightly higher hydrogen ion concentration than that of the cortex. The iso-electric point of the nucleus is at p_H 5.44 and that of the cortex at p_H 5.10, according to our experiments, which were based on lenses of 1 year old cattle. The work was carried out with the object of throwing some light on the cause of diabetic cataract. From the data published by O'Brien [9] it appears that cataract is frequently associated with diabetes. Of seventy-two unselected cases of cataract in persons whose ages varied between 37 and 87, 28 per cent were associated with distinct signs of diabetes. Also it has been shown (Hertel [10]) that the reaction of the aqueous in persons with advanced diabetes is shifted toward the acid side. This is to be expected from the fact that other parts of the body show a similar change in reaction. Stroebe [11] found that the amount of acetone bodies (acetone, aceto-acetic acid and beta-hydroxybutyric acid) in the fluid of skin blisters, produced artificially by cantharides, was directly proportional to the gravity of the disease and paralleled that in the blood. Moreover, it has been shown by Bülow-Hansen [12] that in severe diabetes the spinal fluid, which resembles the aqueous, becomes charged with acetone bodies more readily than the blood. The acetone bodies in the spinal fluid as compared with those in the blood are also less accessible to the influence of insulin. The injurious effect of the acetone bodies, according to the same author, consists in (*a*) the inhibition of the activity of peroxidases, (*b*) the hemolysis of the blood and (*c*) the precipitation of the proteins of the blood. Some of our unpublished data show that the hydrogen ion concentration of the normal lens is about 7.40. Thus approximately only 2 p_H units intervene between the normal reaction of the lens and the iso-electric point of its protein. From the tables it is seen that as soon as the crystalline structure of the lens is destroyed, some of the lens protein comes down as a (white) precipitate even at a normal p_H. It is general knowledge among investigators in this field that in conditions characterized by acidosis, such as diabetes (Kochmann-Römer [13]), eclampsia (Imre [14]) and hyper-

9. O'Brien, C. S.: Biochemical Studies of the Blood in Patients with Senile Cataract, Tr. Am. Ophth. Soc. **76**:438, 1928.

10. Hertel, E.: Ueber die Bestimmung der Wasserstoffionenkonzentration im Kammerwasser, Arch. f. Ophth. **105**:421, 1921.

11. Stroebe, F.: Ueber die Verteilung der Ketonkörper zwischen Blut und Hautblaseninhalt beim Diabetes mellitus, Ztschr. f. klin. Med. **112**:312, 1930.

12. Bülow-Hansen, V.: Ueber das Verhalten der Ketonkörper in Blut, Urin und Spinalflüssigkeit unter besonderer Berücksichtigung des Coma diabeticum, Acta med. Scandinav. **72**:399, 1929.

13. Kochmann, M., and Römer, P.: Experimentelle Beiträge zum pathologischen Flüssigkeitswechsel des Auges, Arch. f. Ophth. **88**:528, 1914.

14. Imre, J., Jr.: Die regulatorische Wirkung der endokrinen Drüsen auf den intraokulären Druck, Arch. f. Augenh. **88**:155, 1921.

pituitarism (Imre[14]), the intra-ocular tension is often decreased. According to the work of Poos[15] and Wiechmann,[16] there exists an inverse relationship between intra-ocular pressure and the amount of blood sugar. They ascribe the lowered intra-ocular pressure to dehydration of the tissues. This may be brought about by the diuretic action of the accumulated sugar. There is reason, therefore, to suspect some slight shrinkage in the lens fibers as a result of this dehydration. If such a condition persists for a long time, formation of opacities may be brought about by at least two mutually operative causes: (1) damage to the crystalline structure and (2) precipitation of the protein by acetone bodies. The acetone bodies are said to be more effective than mineral acids in precipitating the protein (Watanabe[17]); they decrease the surface tension and are therefore more readily adsorbed by the protein (Bülow-Hansen[12]).

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15. Poos, F.: Klinische Untersuchungen über Beziehungen zwischen Osmose, Blutdruck und Augendruck: I. Hypertonie durch Senkung des Blutzuckerspiegels bei jugendlichem Diabetes und Glaukom mit Diabetes, Klin. Monatsbl. f. Augenh. **84**:340, 1930.

16. Wiechmann, E.: Klinische Untersuchungen über die Beziehungen zwischen Osmose, Blutdruck und Augendruck, Klin. Monatsbl. f. Augenh. **85**:815, 1930.

17. Watanabe, W.: Ueber das Puffervermögen des Glaskörpers und des Kammerwassers, Acta Soc. ophth. Jap. **34**:473, 1930; abstr., Zentralbl. f. d. ges. Ophth. **24**:22, 1930.

ROENNE'S NASAL STEP AS STUDIED WITH STIMULI OF DIFFERENT VISIBILITIES*

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One cannot fully test the soundness of the ice on a skating pond or river by applying pressure at one spot. Similarly we cannot fully know the fundamental condition of the retina and the sensorium as a whole by applying our tests at one or only a very few points in the visual field. In preliminary studies, at least, a broad survey must be made for the various types of disease to find the characteristic localities of disturbance. When these are known, the procedure to be used in routine examination can, it is hoped, be greatly simplified.

In the determination of the limits of sensitivity, for example, one of the ways of broadening our survey is by using a series of stimuli differing in visibility. With stimuli of medium and high visibility the more remote parts of the field are tested, and with stimuli of low visibility the more central regions. From this it is obvious that with stimuli of low visibility disturbances in the more remote portions of the field cannot be detected because these stimuli are not sensed in that region even by normal eyes; and that with stimuli of medium and high visibility disturbances in the more central portions of the field are not so readily detected and often cannot be detected at all even by a trained observer.

The course of development of the nasal step presents a somewhat unique case of the need for the use of stimuli differing in visibility. From general considerations of the phenomenon several possibilities are suggested as to the location of the beginning of the loss of sensitivity and as to the course of its spread.

1. The step makes its first appearance in the more remote parts of the field. At this stage of development an indentation in the field at or near the raphé could not be detected with a stimulus of low visibility, because, as already stated, these stimuli are not sensed that far out in the field. As the disturbance progresses, the depth of the indentation may be decreased or the step may be entirely leveled off by a compensating

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contraction in the opposite side of the raphé. At a later stage of development, inequality of contraction may begin again and the step reappear. At least this is the course of development that is indicated by the use of stimuli graded as to visibility. That is, in one and the same examination we find in some cases a step with a stimulus of high visibility, a decrease in the depth of the indentation due to a decrease in the relative amounts of the contraction above and below the raphé with a stimulus of lower visibility, a complete disappearance of the step with a stimulus of still lower visibility and the reappearance of the step with a stimulus still further reduced in visibility. In other groups of cases this is only indicated as a schematized or composite picture of the results obtained in several cases. In this theory of the origin and course of development of the loss of sensitivity, the step is considered as a phenomenon of contraction of the field.

2. The step begins as an impairment within the field of sensitivity, not at its limits or border. In this case the normal limits of the field would be given by stimuli of high visibility, or at least no indentation would be obtained. The steplike indentation of the limits could be obtained only with some stimulus or stimuli of lower visibility. The only evidence of the local impairment of sensitivity that could be expected with stimuli of higher visibility would be a possible dimming of the sensation aroused in the region of the defect. This view would make of the step a kind of scotoma. The possibility or strong probability of a connection with the Bjerrum scotoma is obviously implied in this view.

3. In some cases the step is a phenomenon of contraction of the field and in others, in the incipient stages at least, a scotoma. In still other cases it may begin as a depression of sensitivity which occurs nearly simultaneously over a large part of the upper or lower nasal halves of the field or in more than one part of these halves of the field; that is, if a contraction, it is not a plainly marked or gradual contraction.

With reference to the aforementioned possibilities of explanation it may be noted that the absence of a step with 1 degree white on black as a stimulus, which is seen out to the far periphery, and its presence with a stimulus of lower visibility, which has its limits in the midperiphery, do not argue necessarily that there is an impairment of sensitivity at the limit of the stimulus of lower visibility and not at the limit of 1 degree white on black; in other words, that the loss of sensitivity is scotomatous in origin. Two reasons may be offered for this statement: (*a*) There may have been earlier a contraction of the field above or below the raphé for a 1 degree stimulus which was later compensated for by an equivalent contraction on the other side of the raphé, and (*b*) the sensitivity gradient for form stimuli is steep in the far periphery and very gradual in

the midperipheral and paracentral portions of the field. Because of this, a very marked disturbance must be present to narrow the limit by a detectable amount for a stimulus of high visibility, while a comparatively small disturbance in function would be found to narrow the limit for the stimulus of lower visibility. For example, for a nonpathologic eye in a typical case, the limit for 1 degree white on black (coefficients of reflection respectively 78 and 4 per cent) under 7 foot-candles of illumination at the horizontal raphé in the nasal field was 64 degrees; for 1 degree white on Hering gray no. 13 (coefficients of reflection respectively 78 and 15 per cent) the limit was 63 degrees. In the first case there was a 74 per cent difference in the coefficient of reflection between stimulus and background, and in the second case, 63 per cent, a change of 11 per cent. Both of these were stimuli of high visibility. In order to narrow the limit by 1 degree in this part of the field, then, the actual difference in the amount of light reflected from stimulus and background had to be decreased by 0.77 foot-candle. This shows that 1 degree white on black is a superthreshold stimulus at its experimentally determined limit; that is, a considerable depression of sensitivity is required to change its limit by a detectable amount.

The situation is quite different, however, with stimuli of low visibility. For example, the limit for 1 degree white on Munsell gray no. 8 (coefficients of reflection respectively 78 and 64 per cent) at the horizontal raphé in the nasal field was 44 degrees; for 1 degree white on Munsell gray no. 7 (coefficients of reflection respectively 78 and 69 per cent), 30 degrees. In the first of these cases there was a 14 per cent difference in the coefficient of reflection between stimulus and background, and in the second 9 per cent, a change of only 5 per cent. In this case a decrease in the difference in the amount of light reflected from stimulus and background of only 0.35 foot-candle narrowed the limit by 14 degrees. It is thus seen, as already noted, that a very small disturbance in function is sufficient to narrow the limits to a detectable amount when stimuli of low visibility are used.

A special study of these points in relation to the location of the beginning of the loss of sensitivity in case of the nasal step will be made in later work. In the present study we have been concerned only with finding more sensitive and comprehensive ways of detecting the indentation, when present, at all distances from the center of the field. There is, without doubt, comparatively little chance of detecting the step, particularly in the incipient or very early stages of glaucoma, if only a 1 degree form stimulus (white on black) or stimuli of only one order or degree of visibility are used. At least a much more adequate and comprehensive examination can be made with stimuli representing a variety or series of visibilities. Greater importance would perhaps be ascribed

to the presence of the indentation as a diagnostic sign in glaucoma, had it been the practice or custom to use a variety of stimuli in making the examination above and below the raphé. In any event, if it is felt by the examiner that the test for the step is important to the diagnosis of any particular case, the procedure to be recommended is, by all means, that the limits above and below the raphé for the required distance be determined with stimuli of more than one order of visibility.

As the study of the visual field in relation to diagnosis proceeds, the need for organized, systematic research becomes more and more clearly evident. This research must, of necessity, be detailed. That the mass of detail may become somewhat discouraging to the busy practitioner is only natural. From it, however, will come eventually a system of examination that will save time in diagnosis, rather than consume it unduly; that is, with a cleancut, well planned, confident procedure, more can be accomplished in one examination than could be accomplished otherwise with a number of examinations. This will be time-saving both to the patient and to the examiner, and will lead to an earlier and more certain diagnosis.

METHOD OF WORKING

The cases were studied by means of the Ferree-Rand perimeter and its auxiliary tangent screen. The illumination of the arc of the perimeter and of the tangent screen in the meridian under investigation was kept constant at 7 foot-candles. In searching for the presence of a nasal step with the perimeter the procedure employed was as follows: The limit of sensitivity to the stimulus used was first determined by the usual preexposure method in the nasal half of the horizontal meridian and then alternately in several positions above and below this meridian, such as 3, 6, 9 or 15 degrees, the examination being continued until the presence or absence of an indentation was ascertained. While these limits were being determined, the observer was cautioned to keep his head as motionless as possible since any slight movement of head or eye tended to make the determinations uncertain and inaccurate. With many subjects a consistently differential result could not be obtained closer than 6 degrees above or below the horizontal meridian, and in some cases not closer than 15 degrees. In order further to outline the indentation the limits were usually obtained 15 and 30 degrees above and below the horizontal meridian, as well as the customary 45 degrees, and in still a greater number of meridians if the results warranted closer study. In the maps presented in this paper the additional meridians investigated are indicated, and the limiting points are connected by straight lines.

In cases in which the fields were contracted within the 25 or 30 degree circle, the tangent screen of the perimeter was used to outline the boundaries. To determine whether there was a nasal step in these cases,

the limits were determined above and below the horizontal meridian at sufficiently small intervals. In determining these limits the stimulus was moved from without toward the center of the field along the radii of the screen. The reason for moving in this instead of the reverse direction or in both directions was discussed at length in a previous paper.[1] As a guide for the movement, radial lines are stamped on the surface of the screen at intervals of 15 degrees. These lines are sufficiently distinct to be seen by the examiner, but they cannot be seen by the patient. The stimulus is moved along these lines and between them at points where minuteness of examination is required. When a break is discovered, the step or indentation is outlined by moving the stimulus from the blind to the seeing area along a series of lines at right angles to the horizontal meridian. These lines are spaced at intervals of 5 degrees or less, as the situation may require. The point at which the stimulus is seen is lightly marked on the tangent screen with white or colored chalk and later transferred to the permanent record.

The following stimuli were used to determine the outlines of the fields: one stimulus of high visibility (1 degree white on black), two stimuli of medium visibility (1 degree white on gray and 0.5 degree white on black) and three stimuli of low visibility (the form stimulus 0.17 degree white on black and two color stimuli, 1 degree blue and 1 degree red on grays of the approximate brightness of the colors at their points of disappearance in the periphery). The color stimuli used were obtained from blue and red pigments of the Heidelberg series. The coefficient of reflection of the gray background on which they were viewed was 9.6 per cent. The coefficient of reflection of the form stimuli was 78 per cent; that of the black background on which they were viewed, 3.5 per cent, and that of the gray background, 19 per cent. At the distance of the perimeter arc and the tangent screen (33 cm.), the sizes of stimulus for 1, 0.5 and 0.17 degrees were respectively 5.8, 2.9 and 1 mm.

For the sake of comparison it is desirable that all of the stimuli provided should have been used in each case. With clinical cases this is not always possible. The patient cannot always be secured for an adequate length of time at one sitting, and frequently a return sitting cannot be arranged. Because of these difficulties, a great deal of our data, though instructive, cannot be used for publication, and the comparisons for the cases presented are not always as complete as we should like to have them.

Since neither diagnosis nor the presentation of cases is the purpose of this paper, space will not be taken for the inclusion of all the medical and clinical data that are available for each case. We are interested

1. Ferree, C. E.; Rand, G., and Sloan, L. L.: Sensitive Methods for the Detection of Bjerrum and Other Scotomas, Arch. Ophth. **5**:224 (Feb.) 1931.

only in showing the need for a series of stimuli graded as to visibility for the detection of the nasal step and to throw such light as we can on the development of this pathologic sign.

RESULTS

The results selected for presentation are shown in charts 1 to 8. They are typical of the results obtained with the stimuli employed, covering a much larger number of cases. It has been the aim in the presentation to select from the records at our command examples of various types and various stages of development of the pathologic sign known as the nasal step, and to group these when possible as to type of step, separating when desired the maps for the right and left eye of an individual. Chart 1 shows the progress of a nasal step toward an upper hemianopia as mapped with stimuli of different visibility; chart 2, two cases having more pronounced steps for color than for form stimuli; charts 3 and 4, cases in which steps occur for stimuli of high visibility and almost hemianopia for stimuli of low visibility; chart 5, cases in which the outline of the limits shows a series of fairly regular steps or terraces as if the contraction had taken place in concentric rings; chart 6, two unusual types of nasal step; chart 7, two cases in which the step occurs in conjunction with a very marked general contraction of the field, and chart 8, nasal steps mapped on successive dates. The blind spot and scotomas mapped with certain stimuli are included in the field charts for the purpose of showing their relation to the cuts and indentations found in the outlines of the fields. The data found on these scotomas are not, however, treated in the same detail as was done in an earlier paper.[1] As was stated in the preceding section, only a brief presentation of medical data will be made for each case, summarized from the data available.[2]

CASE 1 (charts 1 and 6 *B*).—Miss E. I., aged 55, had chronic bilateral glaucoma simplex. She first came under observation on Sept. 27, 1930. At that time the tension in the right eye was 35; that in the left eye, 25.[3] The right disk appeared cupped; the left was not plainly seen. Vision in the right eye was 20/15—1 with a +1.25 sphere; in the left eye, 20/15—3 with a +1.50 sphere and a +0.37 cylinder, axis 150 degrees. Pilocarpine was prescribed. Under this treatment the tension was high normal, but rose to 41 in the right eye and 37 in the left when the pilocarpine was discontinued for eight hours. A bilateral corneoscleral trephine was performed on December 18.

An examination was made for the nasal step on Oct. 14, 1930. The data for the left eye only are shown in chart 1. The data for the right

2. These data were taken from the records of the Wilmer Institute. Dr. W. H. Wilmer and the members of his clinical staff gave us their assistance.

3. All values of intra-ocular pressure are given in terms of the revised Schiötz scale.

eye, because of the difference in the type of result obtained, are shown in connection with that of another case in chart 6. For the left eye the following stimuli were used: 1 degree white on black, 1 degree white on gray (coefficient of reflection 19 per cent), 0.5 degree white on black, 0.17 degree white on black and 1 degree red and 1 degree blue on gray of the brightness of the color. For 1 degree white on black no step was found. For the remainder of the stimuli steps were found. The cut in this case was in the upper half of the field. It took the form of an abrupt indentation near the horizontal meridian which, in general, became not only deeper, but widened out toward an upper hemianopia as stimuli of lower visibility were used. For 1 degree white on gray

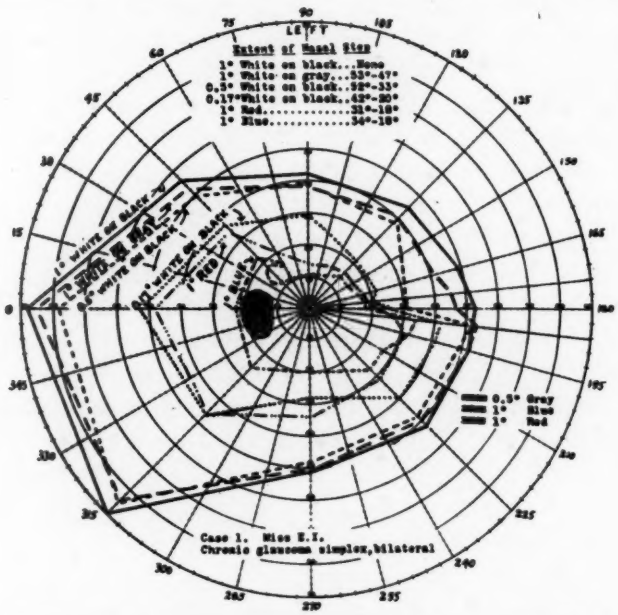

Chart 1.—The nasal step as studied with stimuli of different visibility. No step was found with 1 degree white on black. With stimuli of lower visibility steps were found which increased in depth as the visibility of the stimulus was decreased, and broadened out toward an upper hemianopia. With 1 degree red and blue the blind spot was enlarged and showed slight scotomatous extensions above and below, but there was no detectable connection of the scotoma with the nasal step.

the horizontal indentation extended from 53 to 47 degrees; for 0.5 degree white on black, from 52 to 33 degrees; for 0.17 degree white on black, from 42 to 20 degrees; for 1 degree red, from 31 to 18 degrees, and for 1 degree blue, from 34 to 18 degrees. The most significant effect of the use of stimuli of lower visibility, however, was not on the depth of the cut but in the range of contraction in the upper half of the field. For 1 degree red and blue, for example, the field was lopped off

over a range extending from the 180 degree around to the 45 degree meridional quadrant. The prognostic indication here is plainly an upper hemianopia. The loss of sensitivity, too, is from the periphery inward. In chart 1 are shown also maps of the blind spot for 0.5 degree gray (coefficient of reflection 19 per cent) on black, 1 degree red and 1 degree blue on gray of the brightness of the color. These maps are designated in accordance with the key shown in the chart. The map for 0.5 degree gray on black falls within the normal range of sizes; those for 1 degree red and blue, however, are enlarged. The enlargement is greater for blue than for red and shows a slight scotomatous extension above and below.

For the right eye (chart 6 *B*) 1 degree white on gray (coefficient of reflection 19 per cent), 0.5 degree white on black and 0.17 degree white on black were used as stimuli. For 1 degree white on gray a step of a depth of 15 degrees was found; for 0.5 degree white on black the depth of the step was only 5 degrees. For 0.17 degree white on black a wedge-shaped indentation was found with an angular spread of 30 degrees fairly equally distributed above and below the horizontal meridian. The sides of this indentation were of unequal length, 13 degrees above and 21 degrees below. The blind spot was mapped with 0.5 degree and 0.17 degree white on black. For 0.17 degree white on black there was enlargement and a broad scotomatous extension above to 5 degrees beyond the fovea. For 0.5 degree white this scotoma was narrower and of the shape of a shallow inverted dipper with the handle pointing toward tne blind spot. The lower edge of both scotomas bisected the fovea horizontally. Neither scotoma showed any connection with the nasal steps.

Case 2 (chart 2 *A*).—Mrs. K. G., aged 55, first came under observation on Feb. 6, 1930. She complained of headaches in the temporal and occipital regions; her vision was slightly blurred at times and there was redness and inflammation of the right eye. Ophthalmoscopic examination of the right eye showed many vitreous opacities, and a slight cupping and pallor of the disk at the lower temporal side. The rest of the disk was of good color. The left eye showed a general cupping of the disk, good color and a slightly blurred margin. The tension in both eyes was 20. Dilation of the pupil produced no increase of tension. Vision in the right eye was 20/15 with a +0.50 sphere, —2.50 cylinder, axis 95 degrees; in the left eye, 20/15 with a —1.12 cylinder, axis 75 degrees. Pilocarpine was prescribed on February 6. Under its action the subjective symptoms disappeared.

An examination was made for the cut in the nasal field on Feb. 11, 1930. The stimuli used were 1 degree white on black, 1 degree white on gray (19 per cent coefficient of reflection), 1 degree red and 1 degree blue on a gray of the brightness of the color. The results for the right eye only will be given in this paper. No cut was found for 1 degree white on black and only a slight cut for 1 degree white on gray. Well marked cuts were found, however, for 1 degree red and 1 degree blue.

The cut for the former extended from 25 to 13 degrees, and for the latter from 26 to 13 degrees. A progression toward an upper hemianopia was also shown for both colored stimuli, greater for the red than for the blue. The blind spot was mapped for 0.5 degree white on black, 0.17 degree white on black, 0.5 degree gray (19 per cent) on black, and 1 degree red and 0.5 degree blue on gray of the brightness of the color. It was enlarged and showed scotomatous extensions both above and below for all of these stimuli except 0.5 degree white on black. The enlargement and scotomatous extensions for red and blue were several times greater than for any of the form stimuli. There was no detectable connection between these scotomas and the nasal steps.[4]

CASE 3 (charts 2*B* and 3*A*).—Mrs. K. Y., aged 66, had bilateral glaucoma simplex. The case had been diagnosed as glaucoma, and treatment with pilocarpine

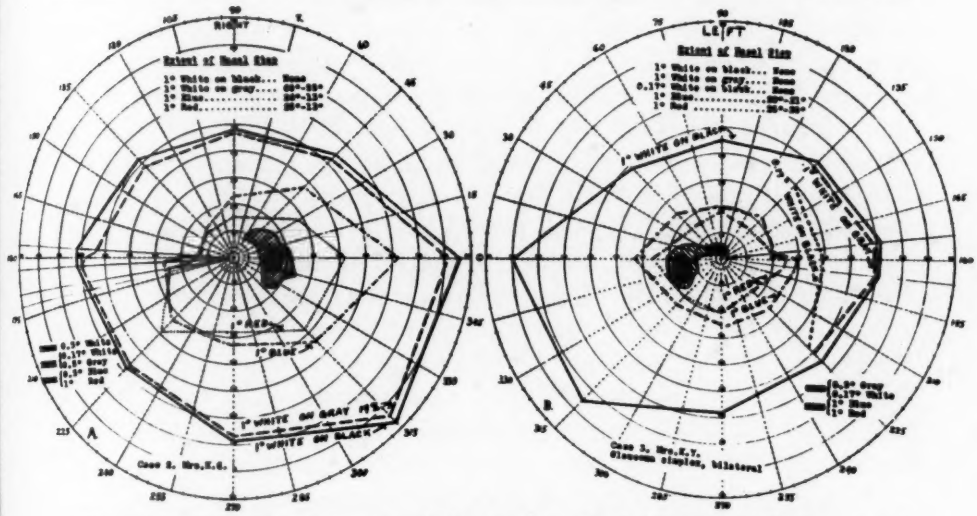

Chart 2.—The nasal step as studied with stimuli of different visibility. In *A*, well marked steps which broaden out toward an upper hemianopia were found with color stimuli; none was found with 1 degree white on black; and but a slight indentation with 1 degree white on gray (coefficient of reflection 19 per cent). In *B*, also, a step was found with color, but none with any of the form stimuli. With the form stimulus of low visibility, however, there was a nasal contraction which was somewhat greater above than below the horizontal meridian. This compensating contraction may have taken place simultaneously; or it may have taken place successively, in which case there was at some time a nasal step. In both *A* and *B* there was enlargement of the blind spot with scotomatous extensions, but there was no detectable connection of the scotomas with the nasal step.

had been given. When examined here in October, 1930, the outlines of the nerve head of the right eye were clear; there were temporal pallor and atrophic changes, and the disk was cupped on the nasal side. The remainder of the fundus was normal

4. For a more detailed description of the blind spot and scotomas found in this case, see Ferree, Rand and Sloan (footnote 1, p. 234).

with the exception of perivascular changes. The report for the left eye was the same. Tension in the right eye was 33; in the left, 28. Vision in the right eye was 15/100 with a +1.50 cylinder, axis 180 degrees; in the left eye it was 20/20—4 with a +0.62 cylinder, axis 180 degrees. A corneoscleral trephine was performed on the right eye on October 29, and on the left eye on November 11.

The fields for the right and left eye were mapped on Oct. 20, 1930. The stimuli used were 1 degree white on black, 1 degree white on gray (coefficient of reflection 19 per cent), 0.17 degree white on black and 1 degree blue and red on gray of the brightness of the color. The results for the left eye are shown in chart 2 *B*. No steps were found with any of the form stimuli. With the 0.17 degree stimulus, however, there was a nasal contraction which was somewhat greater above than below the horizontal meridian. This compensatory contraction above and below may have taken place simultaneously or it may have occurred successively, in which case there was at some time a nasal step. The contraction in the horizontal meridian was 20 degrees greater for 0.17 degree white on black than for 1 degree white on gray; 45 degrees above this meridian, it was 10 degrees greater, and 45 degrees below only 3 degrees greater. Steps were found for both the red and blue stimuli. The depth of the indentation for red was 5 degrees; for blue it was 8 degrees. The blind spot was mapped for 0.17 degree white on black, 0.5 degree gray on black, and for 1 degree red and blue on gray of the brightness of the color. For all these stimuli the blind spot showed enlargement much greater for color than for form. For the form stimuli there was a detached scotoma just above the fovea. For the color stimuli this scotoma was enlarged and connected with the blind spot. For all the stimuli the nasal contraction showed no detectable connection with the scotomatous enlargements or extensions of the blind spot. For the right eye (chart 3 *A*) there was a slight indication of a step for 1 degree white on black. For 1 degree white on gray, because of a more rapid contraction above the horizontal meridian, the depth of this step was increased to 12 degrees. With 0.17 degree white on black, however, there was no step. The indentation had apparently been smoothed out by some compensating change in the rate of contraction above and below the horizontal meridian. With 1 degree blue all sensitivity had disappeared in the upper half of the field except in a small vertical spur between 20 and 30 degrees on the temporal side and a narrow horizontal strip between 2 and 20 degrees on the nasal side. With 1 degree red only the narrow strip between 2 and 20 degrees remained of the upper half of the field, and the lower half had contracted to about one-half the size of the field for blue. The blind spot was mapped with 1 degree, 0.5 degree and 0.17 degree white on black. For all these stimuli there was enlargement, which was much greater for 0.5 and 0.17 degree white on black than for 1 degree white on black. For 1 degree white on black

there was a narrow, detached arcuate scotoma above, which extended from near the blind spot to the horizontal meridian on the nasal side near the fovea. For both 0.5 and 0.17 degree white on black the scotoma was enlarged and connected with the blind spot. The enlargement for 0.17 degree white was very great, involving the fovea and a considerable area both above and below the horizontal meridian on the temporal side. There was no detectable connection of these scotomas with the limits of the field for the form stimuli.

CASE 4 (chart 3 *B*).—Mrs. B. I., aged 73, had bilateral glaucoma simplex with incipient cataracts. She had complained of smarting in the left eye for the past two years. There was also impairment of vision in this eye, more marked for distance. Ophthalmoscopic examination of the right eye showed shallow cupping

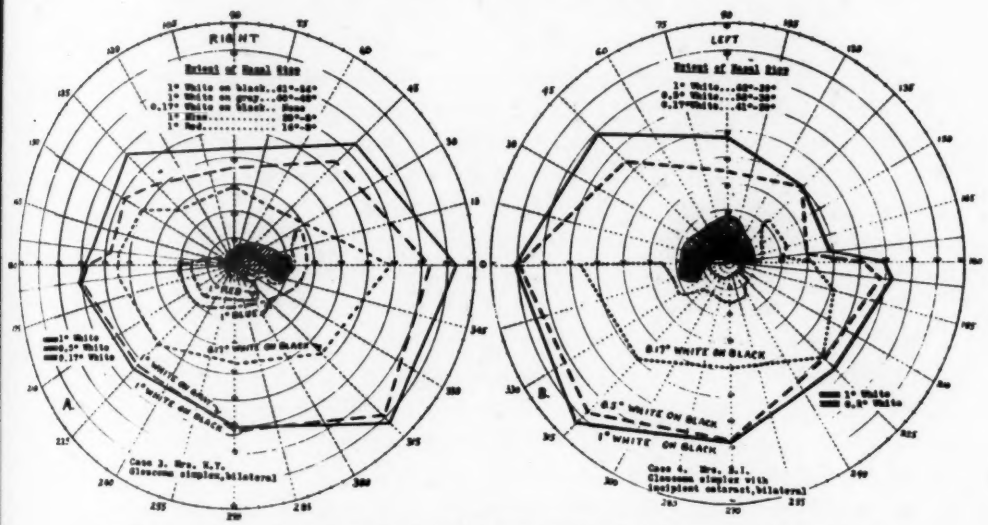

Chart 3.—The nasal step as studied with stimuli of different visibility. In both *A* and *B* steps were found for stimuli of both high and low visibility. With stimuli of low visibility, however, the disturbance had developed into an upper hemianopia. In *A*, a very slight indentation was found with 1 degree white on black and a considerably greater one for 1 degree white on gray, 19 per cent. With 0.17 degree white on black the indentation had been smoothed out by a compensating contraction below the horizontal meridian. With the color stimuli, sensitivity in the upper half of the field was almost entirely lost. In *B*, the nasal indentation was deep for 1 degree white on black and still deeper for 0.5 degree white on black. For 0.17 degree white on black the upper field was almost entirely gone. There was also a broad arcuate indentation below the fovea which had the appearance of a scotomatous extension of the blind spot. Not only is the course of development of the disturbance shown by the results with these three stimuli, but also the comparatively great rapidity with which the disturbance is spreading. In *A*, there was a scotomatous extension of the blind spot above with 0.5 degree and 0.17 degree white on black, and a detached scotoma with 1 degree white on black. These scotomas, however, showed no detectable connection with the nasal steps for the form stimuli. In *B*, there were scotomatous extensions above for both 1 degree and 0.5 degree white on black, which at places grazed the limits of the field for 0.17 degree white on black.

of the disk with a broad halo. The color of the nerve was good with the exception of a slight paleness at the temporal side. For the left eye the appearance of the nerve head was the same as for the right. There were no other lesions in the fundus. The examination was rendered difficult by nystagmoid movements. Tension in the right eye was 35.5, and in the left eye, 33.5. Vision in the right eye was 20/15—1 with a +0.25 sphere, +1.25 cylinder, axis 175 degrees; in the left eye, 20/20—3 with a +1.25 cylinder, axis 180 degrees. A corneoscleral trephine was performed on the left eye on March 3, 1931.

The fields were mapped for the left eye on March 2, 1931. Three form stimuli were used: 1 degree, 0.5 degree and 0.17 degree white on black. Deep nasal steps were found for the first two of these. For the 1 degree stimulus the indentation broadened out toward an upper nasal quadrant anopsia. For the 0.5 degree stimulus, there was considerable contraction of the whole upper half of the field. For the 0.17 degree stimulus the entire upper half of the field was blind with the exception of a small spur from 10 to 20 degrees on the nasal side of the fovea. There was also a broad arcuate indentation below the fovea extending from the horizontal meridian around the fovea nearly to the horizontal meridian on the nasal side. This indentation had the appearance of a scotomatous extension of the blind spot. In this case there was apparently a slight depression of sensitivity over the entire upper half of the field, spreading into the lower half around the fovea. This depression was the greatest near the horizontal meridian on the nasal side and apparently began there. The course of spread and relative amounts of depression are clearly indicated by the limits with the three stimuli graded as to visibility. The results with these stimuli show not only the course of spread but the comparatively great rapidity with which the depression is developing. The blind spot was mapped with 1 degree and 0.5 degree white on black. For both of these stimuli there was enlargement with broad scotomatous extensions above, which passed around the fovea to the horizontal meridian on the nasal side. The scotoma for 1 degree white on black was in places about 10 degrees wide; for 0.5 degree white on black, nearly 20 degrees wide. Neither showed any connection with the nasal contraction of the field for the same stimuli. For 0.17 degree white on black, however, the contraction of the field seemed to have merged with the scotomatous extensions of the blind spot.

Case 5 (chart 4 *A*).—Mr. T. F., aged 41, had chronic glaucoma simplex in the right eye and secondary cataract in the left eye. His case came under observation in December, 1925. Vision was completely gone in the left eye, and there was a history of failing vision in the right eye for one year. From 1925 to 1929 several operations were performed which did not succeed in reducing the tension to normal limits. On Jan. 2, 1930, the tension was 30. The disk was cupped and pale. Vision was approximately 7/40.

The fields were mapped on January 22. The stimuli used were 1 degree white on black, 1 degree white on gray (coefficient of reflection 19 per cent) and 1 degree blue on gray of the brightness of the color. A slight indentation above the horizontal meridian was found with 1 degree white on black. A change to 1 degree white on gray caused a contraction of nearly 25 degrees in the horizontal meridian, with compensating contractions above and below to give a very shallow triangular indentation with a broad base. In this area great susceptibility to fatigue was shown to stimuli of higher visibility. For both form stimuli the upper half of the field showed considerable contraction. With 1 degree blue a much more rapid progress was shown in the contraction above than below the horizontal meridian, amounting to the loss of almost all

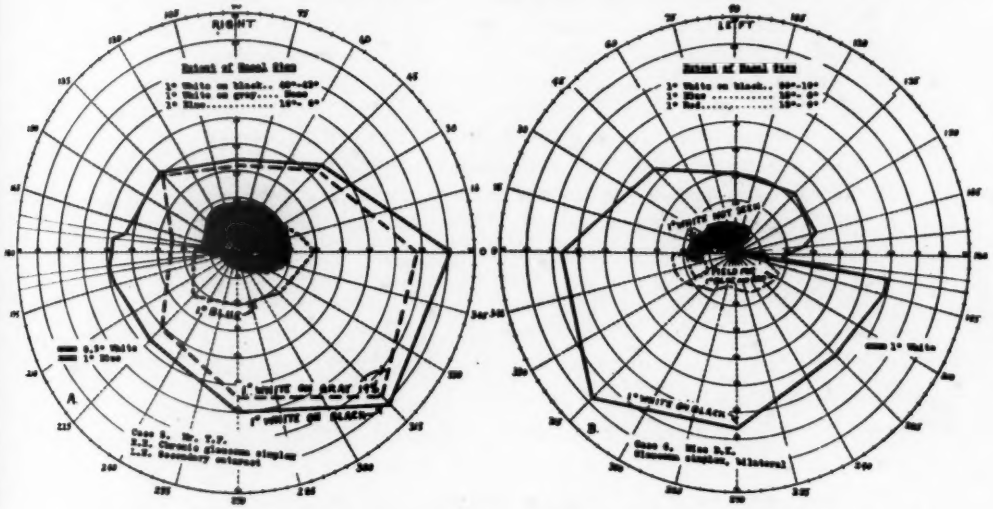

Chart 4.—The nasal step as studied with stimuli of different visibility. In *A*, a slight indentation above the horizontal meridian was found with 1 degree white on black. A change to 1 degree white on gray (19 per cent) caused a contraction of nearly 25 degrees in the horizontal meridian with compensating contractions above and below to give a very shallow triangular indentation with a broad base. The upper half of the field was contracted for both stimuli. One degree blue also showed a much more rapid progress in the contraction above than below the horizontal meridian, amounting to the loss of almost all of the upper half of the field. A broad arcuate scotoma was found for 0.5 degree white on black above the fovea, and both above and below for 1 degree blue. In *B*, also, the upper part of the field was contracted with 1 degree white on black, but in this case the contraction involved the temporal field as well. For 1 degree red and blue the entire upper half of the field was blind with the exception of a narrow strip just above the fovea. With 1 degree white on black a broad arcuate scotoma was found above the fovea which extended nasalward to about 35 degrees beyond the middle of the field. It lay entirely within the area blind to 1 degree red and 1 degree blue, but showed no connection with the nasal cut for 1 degree white on black.

of the upper half of the field. The contraction in the lower half of the field, however, was also extensive. The blind spot is shown as mapped with 0.5 degree white on black and 1 degree blue. For both of these stimuli a broad arcuate scotoma was shown above the fovea, and both above and below for 1 degree blue. At places the scotoma was nearly 20 degrees wide. For 0.5 degree white on black the scotoma extended from the blind spot around the fovea above to the horizontal meridian on the nasal side, and for 1 degree blue it completely encircled the fovea. These scotomas were both coextensive with a part of the area blind to blue.[5]

CASE 6 (charts 4 *B* and 8).—Miss D. K., a Negress, aged 51, had bilateral glaucoma simplex. The case came under observation on Jan. 28, 1930. Ten days earlier the patient had noticed inflammation and dimness of vision in the left eye. Both disks were found to be sharply cupped. The vessels were crowded somewhat to the nasal side, especially in the left eye. The tension was found to be around the upper limit of normal. Pilocarpine was prescribed. Examinations made at intervals of two weeks showed the tension to be normal or near the upper limit of normal. A study of the visual fields made on February 28 showed contraction of both form and color fields and a paracentral scotoma. Later studies, made on September 26 and October 3, showed further contraction of both the form and color fields an dan increase in the size of scotoma. A corneoscleral trephine was performed on the left eye on October 17.

The data represented in chart 4 *B* were obtained from the study of the field for the left eye made on Oct. 3, 1930. The stimuli used were 1 degree white on black, and 1 degree blue and 1 degree red on gray of the brightness of the color. With 1 degree white on black the entire upper half of the field was greatly contracted. There was also contraction in the temporal half of the field and a deep sector-shaped nasal indentation, one edge of which was above and the other below the horizontal meridian. The length of the lower edge of this indentation was 40 degrees and that of the upper about 10 degrees. For both 1 degree red and blue (also for 0.5 degree and 0.17 degree white on black, not shown in the chart) there was a complete upper hemianopia. The blind spot for 1 degree white on black was enlarged with a very broad arcuate scotoma above, which extended nasalward to 30 to 45 degrees beyond the middle of the field. There was no connection of this scotoma with the limits of the field as mapped with 1 degree white on black. It lay, however, entirely within the area blind to 1 degree red and 1 degree blue.[6]

CASE 7 (chart 5 *A*).—Mr. S. E., a Negro, aged 58, had glaucoma absolutum in the right eye and glaucoma simplex in the left eye. He came to the clinic for examination on July 1, 1930. He had noticed a dark spot before the left eye three

5. For a description of the scotomas found in this case for other form and color stimuli, see Ferree, Rand and Sloan (footnote 1, p. 244).

6. For a more detailed study of the scotomas in this case, see Ferree, Rand and Sloan (footnote 1, p. 242).

weeks earlier. An operation had been performed on the right eye a year before. In this eye vision was now entirely gone. An ophthalmoscopic examination of the left eye showed a capillary network over the optic disk. The peripheral fundus was normal. The tension was 28. Vision in the left eye was 20/50 without correction.

The fields were mapped on July 1. The stimuli used were 1 degree white on black and 1 degree blue on a gray of the brightness of the color. A deep nasal indentation was found with 1 degree white on black and an extensive contraction of the lower half of the field. The outline of the contracted field took the form of a series of steps or terraces as if the contraction had taken place in concentric rings or zones. There was also some contraction in the upper and temporal parts of the field.

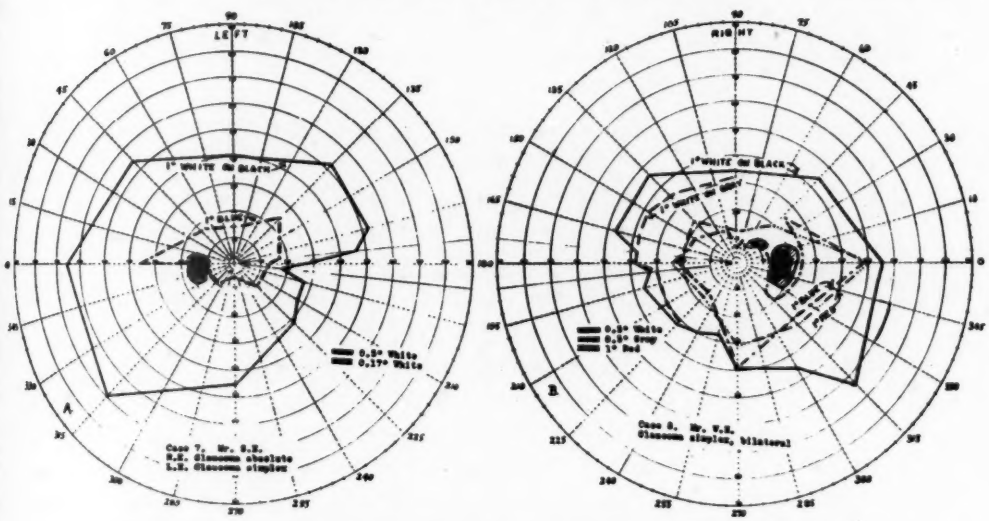

Chart 5.—The nasal step as studied with stimuli of different visibility. In *A*, a deep nasal indentation was found with 1 degree white on black and an extensive contraction of the lower half of the field. The outline of the contracted field took the form of a series of steps or terraces as if the contraction had taken place in concentric rings or zones. With 1 degree blue on gray of the brightness of the color there was extensive contraction of both the upper and lower halves of the field, the contraction of the lower half having progressed almost to complete hemianopia. In *B*, in addition to the nasal indentation there was an extensive contraction of the lower half of the field which also took the form of a series of steps or terraces with 1 degree white on black and 1 degree white on gray, 19 per cent. For 1 degree red and blue there was extensive and irregular contraction of the field in both the upper and lower halves, being more extensive and more irregular in the upper half. In both *A* and *B*, there was an enlargement of the blind spot with scotomatous extensions for the stimuli of low visibility, but there was no detectable connection of these enlargements or scotomatous extensions with the contraction of the fields, except for 1 degree blue in *B*.

With 1 degree blue there was extensive contraction of both the upper and lower halves of the field, the contraction of the lower half having progressed almost to complete hemianopia. The blind spot was mapped with 0.5 degree white on black and 0.17 degree white on black. For the latter stimulus there was considerable enlargement and a slight scotomatous extension below. No connection was shown between this blind spot and the contracting field.

CASE 8 (chart 5 *B*).—Mr. W. H., a Negro, aged 38, had bilateral glaucoma simplex. The patient first came under observation on May 14, 1930. Both pupils were dilated, and there was a moderate injection of the conjunctiva. An ophthalmoscopic examination showed a glaucomatous cupping of the disks with atrophy. This was more marked in the right eye. The blood vessels were crowded to the nasal side of the disk in both eyes. Tension was 60 in the right eye and 55 in the left eye. Pilocarpine was prescribed. The studies of the nasal field were made on May 27 and June 7. At these times the tension in the right eye was approximately 35, and in the left eye 30.

The fields for the right eye were mapped on May 27, 1930, with 1 degree white on black and 1 degree white on gray (coefficient of reflection 19 per cent); on June 7 with 1 degree red and 1 degree blue on gray of the brightness of the colors. There was a marked contraction of the field for 1 degree white on black. On the nasal side this contraction took the form of a series of small steps or terraces beginning at the 165 degree and extending to the 315 degree meridional quadrant. With 1 degree white on gray some of these steps were leveled off by compensating contractions. With 1 degree red and 1 degree blue there was an extensive and irregular contraction of the field in both the upper and lower halves, more extensive and more irregular in the upper half. The blind spot was mapped with 0.5 degree white on black, 0.5 degree gray (coefficient of reflection 19 per cent) on black and 1 degree red on gray of the brightness of the color. For 0.5 white on gray there was considerable enlargement and a detached scotoma above and to the nasal side. For 1 degree red there was a much greater enlargement. There was no detectable connection between these enlargements and the blind areas produced by the contracting fields. The limits of the field as mapped with 1 degree blue, however, seem to indicate a merging with a broad, arcuate scotomatous enlargement of the blind spot.

CASE 9 (chart 6 *A*).—Mr. I. H., aged 63, had uveitis with secondary glaucoma in the right eye; the left eye had been removed because of injury by a piece of steel. The case came under observation on Nov. 28, 1930. Three weeks previous to this the patient had noticed a diminution of vision. About a week later the field of vision became foggy, and pain and anterior headache were experienced. There was no lacrimation or marked photophobia and no haloes were noted. The cornea and media were cloudy (arcus senilis above and below); it was difficult to see the nerve head and fundus clearly. A few normal appearing vessels were seen. The bulbar conjunctiva was slightly injected. The depth of the anterior chamber was

slightly below normal. The pupil was pinpoint in size and fixed. The tension was 41.5. Vision, uncorrected, was 12/200. A cyclodialysis was performed on December 9.

The fields were mapped on December 17. The stimuli used were 1 degree, 0.5 degree and 0.17 degree white on black, and 1.5 degree red and 1.5 degree blue on gray of the brightness of the color. The field for 1 degree white was very greatly contracted with a nasal indentation below of a depth of about 12 degrees and a breadth of about 50 degrees. With 0.5 degree white this indentation was much broader and deeper, and extended within 3 degrees of the fovea. The field also was greatly contracted, particularly in the lower half, almost all of the lower nasal quadrant being entirely blind to this stimulus. For 0.17 degree white on

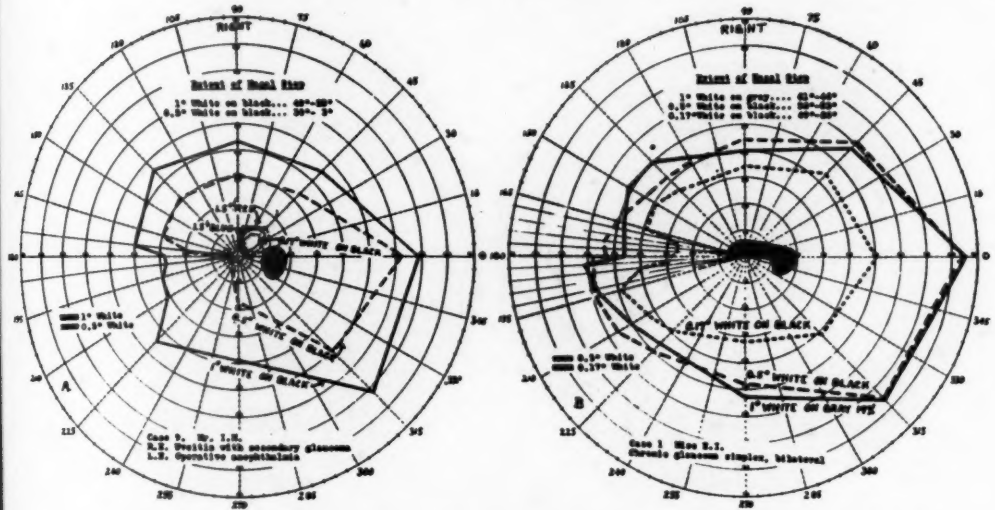

Chart 6.—The nasal step as studied with stimuli of different visibility. In *A*, the fields belong to the class in which deep steps are found with stimuli of high visibility and none at all with stimuli of low visibility; also to the class in which especially great differences are found for stimuli of different visibility, indicating rapid advancement of the disturbance. With 1 degree white on black a step of 12 degrees was found, and with 0.5 degree white on black a step of 27 degrees and loss of sensitivity in nearly all the lower nasal quadrant. Only small patches of sensitivity above and to the temporal side of the fovea remained for 0.17 degree white on black and 1.5 degree red and blue. In *B*, also, the change of the depth and type of indentation was relatively great for the amount of change that was made in the visibility of the stimuli. In *A*, there was a disturbance of the form sense extending from the periphery to the blind spot, as is shown by the results obtained with 0.17 degree white on black. No connection was found, however, between the limits of the contracted field and the limits of the enlarged blind spot for 1 and 0.5 degree white on black. In *B*, no detectable connection was shown between the peripheral disturbance in the form sense and the disturbance around the blind spot.

black there remained only a small patch of sensitivity which was located above and to the temporal side of the fovea. For 1.5 degree blue this patch was somewhat larger, and for 1.5 degree red it was larger still. Little increase in the size of the patch was produced by increasing the size of the colored stimuli to 2 degrees. The eye was entirely blind to 1 degree red and 1 degree blue. The blind spot was mapped with 1 degree and 0.5 degree white on black. There was considerable enlargement for both stimuli, more for the latter than for the former, but the effect around the blind spot was in this case comparatively small as compared with the involvement in the rest of the field. There was no connection between the enlarged blind spot and the limits of the field for the same stimuli.

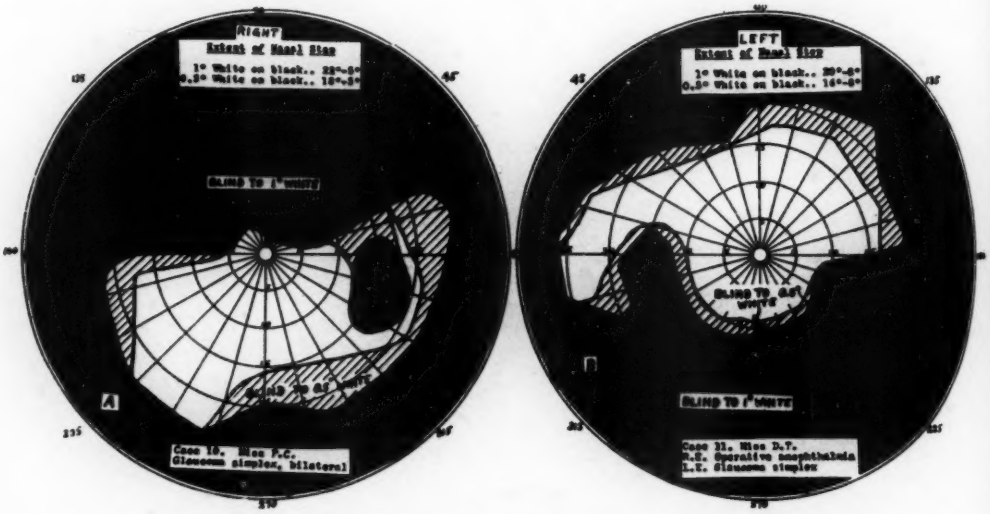

Chart 7.—The nasal step as studied with stimuli of different visibility (tangent screen). In *A*, all but a small portion of the field above the horizontal meridian is lost for 1 degree white on black. For 0.5 degree white on black the area of insensitivity is considerably extended, breaking through from above to the blind spot, or conversely, in the form of an arcuate scotoma. In *B*, the line of nasal indentation follows the horizontal meridian pretty closly to within 7 degrees of the fovea where it seems to join the inner edge of an arcuate scotoma which is connected with the blind spot. With 0.5 degree white on black the field is still further contracted, more from above, however, than from below.

CASE 10 (chart 7 *A*).—Miss F. C., aged 50, had bilateral glaucoma simplex. She complained of impaired vision. The right eye showed pathologic cupping and secondary atrophic changes. The lamina cribrosa was rather prominent. The left eye showed degeneration of the fovea and optic atrophy secondary to intra-ocular tension. Tension in the right eye was 40; that in the left eye, 45. The vision of the right eye, uncorrected, was 20/30; of the left eye, uncorrected, 1/100. The vision of neither eye was improved by correction. A corneoscleral trephine with peripheral iridectomy was performed on Dec. 29, 1930.

The fields were mapped on Sept. 29, 1930. The results are given here only for the right eye. The stimuli used were 1 degree and 0.5 degree white on black. Because of the small fields this examination was made with the tangent screen. For 1 degree white on black almost the entire upper half of the field was gone. Also the lower half of the field was greatly and irregularly contracted. There still remained, however, near the horizontal meridian on the nasal side a steplike formation from 22 to 5 degrees. For 0.5 degree white on black there was a still greater and more irregular contraction of the field, particularly in the lower half. For this stimulus the steplike formation on the nasal side extended from 18 to 5 degrees. The blind spot was mapped with 1 degree and 0.5

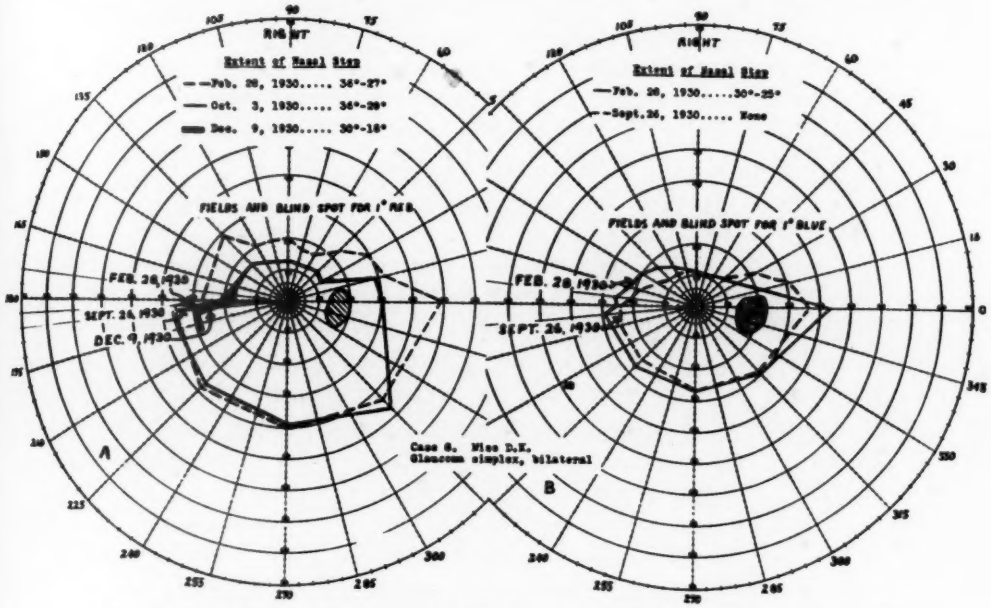

Chart 8.—Changes in the nasal step during a period of time. From Feb. 28 to Sept. 26, 1930, the step was found to become 7 degrees deeper in the left eye for 1 degree red because of contraction above the horizontal meridian and none below. From September 26 to December 9, however, the step had become 4 degrees shallower because the contraction below had progressed faster than the contraction above. In the right eye for 1 degree blue a step was found above on February 28, and none on September 26, because during that period the contraction had been faster below than above. Steps thus may change in depth through a period of time or disappear entirely because of changes in the relative rate of contraction above and below the line of indentation. When only one stimulus is used, a step may be present at one examination and absent at another. When a series of stimuli graded as to visibility is used, there is much less chance of missing this diagnostic sign at any one examination.

degree white on black. For both of these stimuli there was a marked enlargement. For 1 degree white on black there was no connection of the blind spot with the limits of the field. For 0.5 degree white on

black, however, the blind spot was connected by a broad scotomatous extension with the blind area produced by the contraction of the field. Of the cases presented, this is the first in which there has been a connection between the blind spot and the limits of the field for form stimuli of medium visibility.

CASE 11 (chart 7 *B*).—Miss D. T., aged 66, had glaucoma simplex in the left eye; the right eye had been removed. The patient first came under observation on Nov. 12, 1929. She complained of a dimming of vision. There were some deposits on the anterior capsule of the lens; the lens nucleus was somewhat condensed; the outlines of the disk were blurred; there was a deep physiologic cup; the arteries were somewhat tortuous, and there were small yellowish spots on the fundus. The tension was 33. Pilocarpine physostigmine and hot compresses were used, and abscessed teeth were removed. The tension still remained high. A corneoscleral trephine was performed on March 12, 1930. When examined on March 25 and April 5 the tension was normal. On April 24, however, it had risen to 50. The lens was swollen, the cornea was steamy and there were numerous deposits on Descemet's membrane. A cyclodialysis was performed on May 5, and a Lagrange sclerotomy on May 16, the tension remaining low after each operation only for a short time. Vision, with a +2.25 sphere, +0.50 cylinder, axis 120 degrees, was 20/20+3 on Nov. 14, 1929; on Jan. 8, 1930, it was 20/30+1, and on March 12, 1930, 20/200.

The fields were mapped on May 1, 1930. The stimuli used were 1 degree and 0.5 degree white on black. In this case also because of the small fields the examination was made with the tangent screen. For 1 degree white on black nearly all the lower half of the field was gone except an arcuate band around the fovea, about 10 degrees wide, and a broad spur to the temporal side of the blind spot. In the nasal half of the field a steplike formation was found extending from 20 to 8 degrees. The upper half of the field was also greatly contracted. The peculiar shape of the lower edge of the field appears to be due to the fact that the field in contracting merged with a typical Bjerrum scotoma. With 0.5 degree white on black both the upper and lower halves of the field were still further contracted. The nasal step for this stimulus extended from 16 to 8 degrees. The blind spot was mapped with 1 degree and 0.5 degree white on black. For both stimuli there was a great enlargement of the blind spot with broad arcuate scotomatous extensions below, which connected the blind spot with the blind area produced by the contraction of the field. In this case, then, the blind spot was connected with the limits of sensitivity for stimuli of both medium and high visibility. This connection of the blind spot with the limits of the field for stimuli of medium and high visibility occurs apparently only in advanced cases of glaucoma.

It was stated in the introduction that nasal steps may change in depth during a period of time or disappear entirely because of changes in the

relative rate of contraction above and below the line of indentation. In chart 8 are given data to illustrate this point. These data were obtained from examination of the right eye of Miss D. K. (case 6), who had bilateral glaucoma simplex. For some time before and during the period covered by the examinations, the eye was under treatment with pilocarpine. The stimuli used were 1 degree red and 1 degree blue on gray of the brightness of the color. On Feb. 28, 1930, a step of 9 degrees was found above the horizontal meridian for 1 degree red. On September 26 it had become 7 degrees deeper because of further contraction above the meridian and none below, and on December 9, 4 degrees shallower than on September 26 because the contraction below the meridian during this period had progressed faster than the contraction above. For 1 degree blue there was also a step above the horizontal meridian on February 28, but none was found on September 26 because during that period the contraction had been faster below this meridian than above. Steps thus may change in depth during a period of time or disappear entirely because of changes in the relative rate of contraction above and below the line of indentation. More attention will be given in a later paper to the changes that take place during a period of time. The amount of change in the case presented here is not great, perhaps because of the treatment with pilocarpine, but it serves to show the irregular character of the spread of the disturbance. It serves also to show that when only one stimulus is used, the step may be absent at one time and present at another. When a series of stimuli graded in visibility is used, however, there is much less chance of missing this diagnostic sign at any one examination.

SUMMARY AND CONCLUSIONS

1. A series of stimuli graded in visibility is needed for a comprehensive and effective search for the indentations in the limits of the nasal field known as the nasal step. For such a search, tests must be made above and below the horizontal meridian for unequal impairments in sensitivity at suitably spaced intervals from the periphery to the central portion of the field, not at the periphery of the field alone. This can be done with stimuli so graded in visibility as to give limits of sensitivity suitably spaced from the peripheral to the central portion of the field.

2. With 1 degree white on black as stimulus, a step was detected in 67 per cent of the cases presented in this paper; with form stimuli of medium visibility, such as 0.5 degree white on black or 1 degree white on gray (coefficient of reflection 19 per cent), in 90 per cent of the cases; with a form stimulus of low visibility, such as 0.17 degree white on black, in 50 per cent, and with color stimuli, such as 1 degree red and 1 degree blue on gray of the brightness of the colors, in 89 per cent. Of the total number of cases studied, the step was detected with 1 degree

white on black in 57 per cent, with 0.5 degree white on black or 1 degree white on gray (coefficient of reflection 19 per cent) in 85 per cent, with 0.17 degree white on black in 45 per cent and with 1 degree red or 1 degree blue on gray the brightness of the color in 80 per cent.

3. As the disease advances, steps may change in depth through a period of time, disappear entirely and reappear again because of changes in the relative rate of contraction above and below the line of indentation. Because of these irregularities in the contraction both as to time and location of occurrence, the nasal indentation may also show a great variety of shapes at different distances from the center of the field. All these indications as to the course of development which the disturbance takes may in some cases be found in the results of a single examination when stimuli graded in visibility are used; in others they are a schematized or composite picture of the results obtained in several cases.

4. The indentation may occur in either the upper or lower nasal quadrant or in both. In case it occurs in only one of the quadrants it usually widens out toward a quadrant anopsia or hemianopia through a series of irregular contractions. Sometimes the outline of the limits shows a series of fairly regular steps or terraces as if the contractions had taken place in concentric rings. In other cases these steps are very irregular in breadth and shape, but still suggest the spreading of the disturbance in concentric zones or rings. In case the contraction takes place in both the upper and lower quadrants, the limits in some cases take the form of a nearly vertical line, in others that of a shallow indentation with a broad base, and in still others the form of a deep and comparatively narrow indentation with regular or irregular sides of different ratios as to length.

5. From general considerations of the phenomenon several possibilities are suggested as to the location of the beginning of the loss of sensitivity and as to the course of the spread: (*a*) The step first makes its appearance in the more remote parts of the field. At this stage of development an indentation in the field at or near the raphé could not be detected with stimuli of low visibility because these stimuli are not sensed so far out in the field. As the disturbance progresses, the depth of the indentation may be decreased or the step entirely leveled off by a compensating contraction on the opposite side of the raphé. At a later stage of development, inequality of contraction may begin again and the step reappear. In this theory of the origin and course of development of the loss of sensitivity, the step is considered a phenomenon of contraction of the field. (*b*) The step begins as an impairment within the field of visibility, not at its limits or border. In this case the normal limits would be given with stimuli of high visibility; at least no indentation would be obtained. The steplike indentation of the limit could be

obtained only with stimuli of lower visibility. The only evidence of the local impairment of sensitivity that could be expected with stimuli of high visibility would be a possible dimming of the stimulus in the region of the defect. This view would make of the step a kind of scotoma. (*c*) In some cases the step is a phenomenon of contraction of the field, in others, in the incipient stages at least, a scotoma, and in still other cases it may begin as a depression of sensitivity which occurs nearly simultaneously over a large part of the upper or lower nasal halves of the field or in more than one part of these halves of the field. The results obtained in this study seem to indicate a tendency toward two regions of involvement, one in the periphery and the other near the center of the field. The first could not be so successfully detected with 1 degree white on black as with 0.5 degree white on black and 1 degree white on gray (coefficient of reflection 19 per cent) because of its supraliminal intensity, due to the steep gradient of sensitivity in this part of the field. The second could be more successfully detected with colored stimuli than with 0.17 degree white on black because the limits for 0.17 degree white on black were too remote from the center of the field to fall within the region of disturbance in every case. The limits for 0.17 degree white on black apparently fell more nearly between the two regions of disturbance than the other stimuli employed.

6. The sensitivity gradient for the form sense is steep in the far periphery of the field and gradual in the midperipheral and paracentral portions of the field. This difference in sensitivity gradient is one of the reasons why stimuli of high visibility whose limits lie in the far periphery have less sensitivity than stimuli of low visibility for detecting the small local impairments of sensitivity which cause incipient nasal steps. The absence of a step with a stimulus of high visibility and its presence with a stimulus of lower visibility does not necessarily imply, therefore, that there is an impairment of sensitivity at the limit of the latter and not at the limit of the former, in other words, that the loss of sensitivity is scotomatous in origin.

7. No detectable connection of the nasal step with the Bjerrum scotoma is shown in the incipient stages of glaucoma. A connection is found only in advanced stages of involvement of the field. In these stages the inward contraction from the periphery and the outward contraction from the blind spot seem to meet or merge. It is interesting to note, however, that in all the cases reported in this paper, with but one exception, the Bjerrum scotoma, when present, occurred in the same half of the field as the indentation called the nasal step; and in cases in which the scotoma was found both above and below the blind spot, it was more advanced in that half of the field which had the greater contraction from the periphery.

8. In any case in which the search for the nasal step is considered important for diagnosis the recommended procedure is the use of a series of stimuli suitably graded as to visibility in the examination of the limits above and below the horizontal meridian in the nasal field. Very good results can be obtained, however, with form stimuli of medium visibility, such as 0.5 degree white on black or 1 degree white on gray (coefficient of reflection 19 per cent), in conjunction with color stimuli, such as 1 degree red and 1 degree blue on gray of the brightness of the color.

THE METABOLISM OF THE RETINA

FURTHER NOTES *

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As the result of some experiments reported last year,[1] it was concluded that the sugar content of the normal vitreous was always considerably lower than that of the aqueous or blood serum of the same animal. The average blood sugar of cats was found to be 135 mg. per cent. The aqueous averaged 113 mg. per cent, while the vitreous averaged 64 mg. per cent. The samples of aqueous, blood and vitreous were drawn simultaneously, and careful tests were made of the reliability of the methods employed.

I was puzzled how to account for this low vitreous sugar. When the blood sugar of an animal was raised experimentally, the aqueous rapidly equilibrated itself to the new sugar level. The vitreous did so likewise, but being a gel the changes took place much more slowly. This proved, however, that the vitreous could increase its sugar level when the blood sugar increased. Other experiments showed that no diffusion of sugar took place from the anterior to the posterior chambers at normal intraocular pressures. The iris lens contact probably forms a water-tight barrier to the diffusion of fluid backward from the anterior into the posterior chamber. This explains how it is possible for the aqueous sugar to be so much higher than the vitreous and maintain this difference constantly. Were diffusion possible to any great extent, the two would soon equilibrate. A study of the rate at which the various tissues in the eye could split sugar was made at that time, and it was found that the glycolytic activity of the retina was almost double that of any of the other ocular tissues.

This fact seemed to explain the low concentration of sugar in the vitreous. The vitreous is bounded by the retina in practically its whole

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* From the Departments of Physiology and Ophthalmology of the University of Pennsylvania.

1. Adler, Francis Heed: An Investigation of the Sugar Content of the Ocular Fluids Under Normal and Abnormal Conditions, and the Glycolytic Activity of the Tissues of the Eye, Tr. Am. Ophth. Soc. **28**:307, 1930.

extent. Only in its anterior portion is it bounded by other structures, i. e., the ciliary processes and lens. It is, therefore, exposed at all times to the activity of the retina. All substances passing into the vitreous from the blood stream, such as sugar and salts, must come from either the choroidal or retinal circulation. They are, therefore, directly exposed to the activities of the retina in their passage into the vitreous. Under these conditions one should find a lower concentration of sugar in the vitreous than in the aqueous, the latter being enclosed by structures having very little glycolytic activity.

TABLE 1.—*Difference in Sugar Content of the Anterior and Posterior Layers of the Vitreous*

Eye No.	Blood, Mg. per 100 Cc.	Aqueous, Mg. per 100 Cc.	Anterior Vitreous, Mg. per 100 Cc.	Posterior Vitreous, Mg. per 100 Cc.	Difference
1*	284	86	63	53	—10
2*	284	86	81	64	—17
3	108	99	71	44	—27
4	104	73	63	70	+ 7
5	104	83	63	52	—11
6	118	96	65	67	+ 2
7	118	127	64	47	—17
8*	281	158	129	106	—23
9	127	65	42	35	— 7
10	150	..	68	57	—11
11	150	114	88	51	—37

* These animals were anesthetized with ether and chloroform, hence the high sugar values in blood and the discrepancies.

If this explanation is correct, one might expect to find the concentration of sugar in the vitreous not uniform, but exhibiting a gradient with the lowest concentration in the parts close to the retina, and the highest anteriorly toward the iris. This is exactly what was found when the vitreous was frozen, divided into two portions, anterior and posterior, and then analyzed for sugar.

As the figures in table 1 show, the concentration of sugar in the posterior layers of the vitreous is considerably less than that in the anterior layers.

The present experiments were designed to test this hypothesis still further. If this is correct, one should be able to demonstrate a considerable difference in the sugar concentration of the vitreous in the two eyes of an animal when the glycolytic activity of the retina on one side has been impaired or abolished. The sugar concentration should be higher on this side than on the normal.

The integrity of the anatomic structure of the retina is partly necessary in this process of glycolysis; for example, the retinas separated

carefully from the choroid used on an average of 21 per cent of the sugar present in one hour, whereas the corresponding retinas from the opposite eyes ground up with sand used only 11 per cent. Since, even after disruption of the histologic structure, the retinas could still use some sugar a chemical factor seemed also to be concerned, and experiments with a protoplasmic poison, such as sodium fluoride, showed this to be the case. A minute amount of sodium fluoride present in the solution would effectively stop all glycolysis. Obviously neither of these methods were suitable for the present problem.

A few preliminary experiments were done in which a trace of sodium fluoride was injected into the vitreous of one eye in the live anesthetized animal, and twenty-four hours later both eyes were enucleated and the sugar content of the vitreous determined. These experiments were unsatisfactory because of the inflammatory changes which naturally occur in an eye so treated, so that it was felt that this method of experimentation was quite unsuitable.

It is well known that section of the optic nerve results in an atrophy of the retina. An optic atrophy follows a laceration of the optic nerve in the orbit, and degenerative changes take place in the retina. Whether the whole retina atrophies or just certain layers after such an injury is not definitely known.

It was decided, therefore, to produce an atrophy of the retina by cutting the optic nerve and then to study the sugar content of the aqueous and vitreous and the glycolytic activity of the retina. In five cats the left optic nerve was cut just back of the eyeball under ether anesthesia. One month later each animal was killed, and both eyes were removed. The rate of glycolysis of the side on which operation was performed was compared to that of the opposite side. In four of these cats the aqueous and vitreous of the two sides were analyzed for sugar by methods described in my previous paper. In all of the cats the pupil reactions were determined and were found to indicate a lesion of the left optic nerve.

Cats 3 and 4 showed typical pupillary reflexes indicating a complete break in the afferent pathway on the left side, i. e., a prompt direct light reaction but no consensual reaction on the right side. There was no direct but prompt consensual reaction on the left side. The left pupil in cat 5 did not react to light directly or consensually and was widely dilated. The right eye reacted to light directly but not consensually, so there could be no doubt that the afferent fibers on the left side were cut. It is probable that the efferent fibers or the ciliary ganglion on the left side were also damaged at the time of the operation.

In all three cats the retinas on the left side showed atrophy, judged by the gross thickness of this tissue compared with that on the right

side, and by a disappearance of the brilliant green hue of the underlying tapetum. In all three the aqueous sugar and vitreous sugar of the eye on which operation was not performed were normal, whereas the aqueous on the opposite side was higher than normal, and the vitreous very much above normal.

When, therefore, the retina and choroid are atrophic to such an extent that changes in them are visible to the naked eye, the vitreous sugar rises above the normal. The simplest explanation of this is a reduction of the glycolytic activity of the atrophied retina.

A study of the glycolytic activity of the retinas showed this to be the case. The rate of glycolysis of the posterior segments of the normal

TABLE 2.—*Changes in Sugar Content of Aqueous and Vitreous After Optic Nerve Section*

	Aqueous, Mg. per 100 Cc.		Vitreous, Mg. per 100 Cc.	
Cat No.	Control	Experimental	Control	Experimental
3	89	102	59	96
4	83	89	69	153
5	95	125	68	113

TABLE 3.—*Rate of Glycolysis Per Gram of Tissue Per Hour*

Cat No.	Normal Posterior Segment, Mg.	Optic Nerve Cut Segment, Mg.
1	12.8	1.8
3	18.5	14.6
4	5.7	1.7
5	17.7	6.2

eye compared to the eye on which operation was performed was made with the results shown in table 3.

In all four cats the atrophied retinas used much less sugar per gram per hour than the normal side.

It would be of no small physiologic interest to determine what portion of the retina was chiefly responsible for this glycolytic activity.

The utilization of large quantities of sugar is an interesting phenomenon, and at present very little can be said regarding the use that a tissue like the retina makes of this function. Of all the tissues in the body, the retina and brain have the highest rate of glycolysis. Muscle, on the other hand, has a very low glycolytic rate. It is conceivable that tissue like muscle, which can store large quantities of glycogen, burns this for fuel and not the sugar circulating in the blood, whereas the retina and the brain have very little glycogen storage and hence must use the circulating sugar for their physiologic needs.

If in cutting the optic nerve all other structures could be avoided, the damage to the retina might be limited to certain layers, i. e., the nerve fiber and ganglion cell layer. Unfortunately, in cutting the nerve close to the eyeball, blood vessels are unavoidably caught in probably every case, and the resultant atrophy probably involves all of the retinal layers. A series of these retinas are now being examined histologically by Dr. de Renyi of the department of anatomy of the University of Pennsylvania.

In cat 2 of this series, the pupil reflexes one month after operation indicated the usual complete lesion of the left optic nerve, but the retina on this side showed no sign of any gross atrophy. The tapetum looked normal, and the retina itself was of normal thickness.

The aqueous and vitreous showed little difference on the two sides, and the rate of glycolysis of the two sides was alike, i. e., 13.2 and 15.4 mg. per gram tissue per hour for the normal and experimental side, respectively.

This would be easiest to explain by assuming that in this cat enough blood vessels escaped at the time of operation to prevent any retinal atrophy, and hence the two sides were alike. Unfortunately, no histologic preparations were made of these retinas.

I am planning to section the optic nerve in such a way as to spare the blood vessels, and to compare the specimen with those from animals in which the section is made close to the globe and in which the blood supply to the retina is interfered with. Histologic studies will be made of these retinas from which some information may be obtained regarding the layers of the retina that are responsible for the glycolytic activity.

SUMMARY

An atrophy of the retina produced experimentally by cutting the optic nerve in cats results in an increased concentration of sugar in the vitreous. The glycolytic activity of the atrophied retinas is considerably less than that of the normal. These facts confirm the hypothesis previously made that the low concentration of sugar in the vitreous is due to the high rate of glycolysis of the normal retina.

SUBNORMAL ACCOMMODATION *

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The relative infrequency of the occurrence of subnormal accommodation is perhaps the reason it is so easily overlooked. It has been said that the difference between the consultant and the average practitioner in a given field is that the consultant has the ability to recognize and treat correctly the 25 per cent of conditions which the average practitioner does not recognize or treat. Functional insufficiency or subnormal accommodation falls into this small group of difficult cases observed by the ophthalmologist. It is suggested that deficiency of accommodation in young persons would be more often recognized if the profession would adhere more religiously to the taking of the accommodative near point and its interpretation. Refraction is fundamentally an accommodative problem.

Jackson [1] stressed the value of taking the near point of accommodation as a routine, and pointed out that it is often neglected:

> Asthenopia from eye strain usually depends on strain of accommodation. Correction of refractive errors may give relief without any study of accommodation, but one cannot know how much the accommodation is strained without knowing how much of it there is to do the required work. It is neglect of professional duty to prescribe for strain of accommodation on an assumption based on age and general symptoms when we can so easily know just what the accommodation is, and how much can be expected of it. Weakening of accommodation exists and causes annoying and persistent symptoms long before the presbyopic age. . . . The taking of the near point should be as much a part of the routine examination as the use of distance test letters and trial lenses.

Theobald [2] stated: "Subnormal power of accommodation is a not infrequent cause of asthenopia in young people and met with not infrequently in members of the same family . . . a distinct condition only in the young for after middle life it is difficult to distinguish from presbyopia. Headaches and defective near vision are the chief complaints." He held that subnormal power of accommodation was not revealed by determination of the accommodative near point, for

* Submitted for publication, May 30, 1931.
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1. Jackson, Edward: Taking the Near Point, editorial, Am. J. Ophth. **5**:837 (Oct.) 1922.

2. Theobald, Samuel: Subnormal Accommodative Power in Young Persons a Not Infrequent Cause of Asthenopia, Tr. Am. Ophth. Soc. **6**:127, 1891.

patients who have this condition may even hold objects abnormally close. Rather, the defect was evidenced by exophoria of less than 5 degrees in near vision. In other words, if there did not exist a difference in exophoria of from 3 to 4 degrees between distant and near vision, subnormal accommodation was held to exist. There should be a difference of from 3 to 5 degrees in favor of the external muscles in near vision. In four papers on the subject,[3] including 155 cases of the condition, he reported that the incidence was 10 per cent among refraction cases. He regarded the problem as a local one, and treated the condition by addition of plus lenses for near vision and by means of prisms, base out, endeavoring to produce minimal exophoria of 3 or 4 degrees. Huizinga [4] stated that it is often forgotten that the symptoms of deficient accommodation are similar to those resulting from refractive errors. The only way they can be distinguished is to measure the range of the accommodation, a much neglected procedure. A prescription for glasses should not be given until the physician is as familiar with the condition of accommodation as he is with the error of refraction. From two thirds to three fourths of the power can be used without fatigue. Suker [5] reported the condition in association with paralysis of the accommodation. Gould [6] stated that subnormal accommodation or premature presbyopia was particularly troublesome in cases of definite ocular strain in which relief was not obtained by ordinary measures and corrections. He also stated that failure to recognize insufficient or paretic accommodation because of lack of attention to minute details contributed to errors in refraction work. Gould reported twenty-seven cases in which he attributed the condition to local causes and treated the patients by means of bifocal lenses. Establishment of the normal values of accommodation for the different ages was accomplished by Jackson [7] and by Duane,[8] who carried on to completion the original

3. Theobald: Footnote 2; Some Typical Cases of "Subnormal Accommodative Power," Tr. Am. Ophth. Soc., 1894, p. 138; Some Typical Cases of Subnormal Accommodative Power, Ann. Ophth. & Otol. **3**:236, 1894; Some Striking Examples of Subnormal Accommodative Power, Bull. Johns Hopkins Hosp. **24**:282 (Sept.) 1913.

4. Huizinga, J. G.: Necessity for Knowing the Range of Accommodation, Ophth. Rec. **8**:163 (April) 1899.

5. Suker, G. F.: Paralysis and Paresis of the Muscle of Accommodation, Am. J. Ophth. **20**:208, 1903.

6. Gould, G. M.: Subnormal Accommodation and Premature Presbyopia, Am. Med. **9**:103 (Jan. 21) 1905; The Discovery of Subnormal Accommodation in the Young, ibid. **9**:180 (Feb. 4) 1905.

7. Jackson, Edward: The Amplitude and Accommodation at Different Periods of Life, and Its Relation to Eye-Strain, California State J. Med. **5**:163 (July) 1907.

8. Duane, Alexander: An Attempt to Determine Normal Range of Accommodation at Various Ages; Being a Revision of Donders' Experiments, Tr. Am. Ophth. Soc. **11**:634, 1908.

work of Donders.[9] In his report, Jackson analyzed 3,346 cases, giving a table of average, maximal and minimal values for accommodation at different ages, and he stressed the relationship between the accommodative anomalies and eyestrain. "To assume that the accommodative power is calculable by the patient's age is closing our eyes to many obvious or easily obtainable facts." He stated that never since engaging in practice had he prescribed glasses without first measuring the patient's accommodation. He referred to the relative frequency of subnormal values, and reported two cases in patients aged 15 and 19 years, respectively, who required added help for near work.

The question of accommodative values was greatly clarified by the work of Duane [10] in his several papers published between 1908 and 1916. He established tables of values for the accommodation at the various ages. By these he set up certain definite standards by which the normal and the pathologic could be distinguished. He felt that the comparatively little attention paid to accommodative anomalies was due to lack of data on what constitute normal values of accommodation. His table of the minimal, mean and maximal accommodation at the various ages should be a part of the armamentarium of every refractionist. In an outstanding paper on the subject of anomalies of the accommodation, Duane [11] placed the incidence of subnormal accommodation at 5 per cent of all cases in which refraction is done. The symptoms are blurred near vision, inability to do close work, various asthenopic symptoms and conjunctival irritation. Insufficiency differs from paresis or paralysis only in degree. As etiologic factors, eye strain, anemia, neurasthenia, metabolic disturbances, intestinal toxemia, measles, influenza, whooping cough, tuberculosis, nasal obstruction and, possibly, foci of infection were suggested. Treatment consisted of reading glasses, exercise of accommodation and convergence and removal of causes of the trouble. Pilocarpine appeared not to be of benefit. About this time, from 1916 to 1919, focal infection began to be stressed as a cause of lowered accommodative power. Veasey [12] reported six cases of partial or complete loss of accommodation due to focal infection. In five of these

9. Donders, F. C.: On the Anomalies of Accommodation and Refraction of the Eye, London, The New Sydenham Society, 1864.

10. Duane, Alexander: The Accommodation and Donders' Curve and the Need of Revising Our Ideas Regarding Them, Tr. Sect. Ophth. A. M. A., 1909, p. 57; Normal Values of the Accommodation at All Ages, ibid., 1912, p. 683; Studies of the Accommodation, Ophthalmoscope **10**:486 (Sept.) 1912; Importance of Testing Accommodation as a Routine Measure in Refraction Work, New York State J. Med. **13**:666 (Dec.) 1913; Anomalies of the Accommodation, Clinically Considered, Arch. Ophth. **45**:124 (March) 1916.

11. Duane (footnote 10, fifth reference).

12. Veasey, C. A.: Paralysis of Accommodation Due to Focal Infections, Am. J. Ophth. **2**:858 (Dec.) 1919.

cases the cause was in the tonsils, and the condition cleared up after tonsillectomy. One case of sudden loss of accommodative power responded promptly to the draining of an abscessed tooth. In a discussion of Veasey's paper before this society, Duane stated that not only paralysis but also weakening of the accommodative power, subnormal accommodation, must frequently be due to focal infection and also to nasal obstruction. Only recently had infection in the teeth and tonsils come to his attention as causes. He reported one case of accommodative asthenopia that was immediately relieved by tonsillectomy. Holloway [13] reported a case of paresis of accommodation following streptococcus infection from or about catgut sutures after several general operations. Woods [13] called attention to cases in children, in which an irritable ciliary muscle would not relax satisfactorily under any cycloplegic, and in which this spasticity cleared up following the removal of infected teeth. Howe [13] pointed out that spasm can come from focal infection as well as paresis. De Schweinitz [14] expressed the belief that these cases might be due to a diffusible toxin coming from bacteria or otherwise (food poisoning), and that these toxins might find an affinity for certain tissues. White [15] published an account of complete, bilateral paralysis of accommodation from peritonsillar abscess, and recovery following incision and drainage. There was no pupillary involvement. Sumner [16] reported three cases of subnormal accommodation associated with diseased tonsils, in one of which an operation was performed and recovery was prompt. Shoemaker [17] reported two cases in which there was the same difficulty, and his treatment was by pilocarpine and reading glasses. In his last paper on the subject of subnormal accommodation, Duane [18] reported 175 cases observed in twenty years of practice. He included all degrees of lowered accommodative power, from complete paralysis to slight insufficiency. He stated:

> We have more often to do with the less marked and more fugitive varieties of insufficiency which are yet of considerable importance on account of the trouble they occasion. It is not the actual amount of accommodation present but whether that amount is at or below the normal for the age, that determines the symptoms in any given case. Accommodative insufficiency is not absolute but relative. It is said to exist whenever the manifest accommodation is persistently below the minimum for the age of the subject.

13. Discussion, Am. J. Ophth. **2**:880 (Dec.) 1919.

14. de Schweinitz, G. B.: Discussion, Am. J. Ophth. **2**:881 (Dec.) 1919.

15. White, J. W.: Paralysis of Accommodation Following a Peritonsillar Abscess, Am. J. Ophth. **4**:276 (April) 1921.

16. Sumner, Percy: Subnormal Accommodation; the Result of Focal Infection, Am. J. Ophth. **4**:356 (May) 1921.

17. Shoemaker, J. F.: Subnormal Accommodation, Am. J. Ophth. **5**:393 (May) 1922.

18. Duane, Alexander: Subnormal Accommodation, Arch. Ophth. **54**:566 (Nov.) 1925.

This quotation from Duane clearly defines the condition of accommodation that I wish to consider in this paper.

Duane divided accommodative insufficiency into two main groups: (1) static, a condition of inertia, lack of response of the lens or ciliary muscle although innervation is normal, premature presbyopia, and (2) dynamic, innervational, a condition of deficient energy (*a*) with pupillary involvement and (*b*) without pupillary involvement.

This paper is chiefly concerned with the group of dynamic insufficiency without pupillary involvement. Some of the cases, however, clearly fall into the other groups and will be noted as such.

I am presenting here a series of twenty cases of subnormal accommodation among persons of an age unquestionably below that at which presbyopia should normally appear. The picture in these cases is that of reduced accommodative power for the age of the patient, yet definitely short of actual paralysis. Although subnormal accommodation is relative, I believe that it is a definite clinical entity, easily recognizable and of much annoyance to the patient. These cases fall naturally into the classes mentioned: group 1, static accommodative insufficiency, premature presbyopia, and group 2, dynamic insufficiency, with or without pupillary involvement.

REPORT OF CASES

In group 1 there are three cases, as follows:

CASE 1.—A woman, aged 39, complained chiefly of weakness of the eyes for close work; they tired even with the use of glasses. She was wearing a right lens of +1.25 sphere, which gave vision of 6/7 and an accommodative near point of 29 cm., and a left lens of +1.25 sphere, which gave vision of 6/7 and an accommodative near point of 29 cm. The average accommodative near point for her age is 45 cm., and the maximal 40 cm. External examination and casual examination of the visual fields gave negative results. The muscle balance was normal. Manifest refraction of each eye disclosed that +3.00 spheres were necessary for ordinary near vision. Under the influence of a cycloplegic, a +1.50 sphere gave vision of 6/7 in both eyes. The media were clear and the fundi normal, except for slight constriction of the right superior temporal artery. Seven days after the refraction that had been made under a cycloplegic, another examination was made without a cycloplegic. The patient accepted the full correction that had been determined with a cycloplegic; a +1.00 sphere given in a bifocal lens was added to each eye for near work.

The patient's teeth and tonsils had been removed ten years previously. The Wassermann test of the blood was negative. Slight secondary anemia was present. The patient returned after three years and reported that until recently she had been comfortable with the correction given. At the time of her return, however, when she was 42 years of age, she had to hold print away from her eyes to read it, and had headaches after close work. With the correction that has been recorded, vision of each eye was 6/6, and the near point when looking through the part of the lens designed for distant vision was infinity in each eye. The patient held fine print at 58 cm. to read it. External examination, examination of the pupils and

casual examination of the visual fields indicated normal conditions. Examination of muscle balance gave negative results. Refraction after dilatation with cocaine disclosed that a +1.50 sphere gave vision of 6/6 in both eyes. For ordinary range, a +2.00 sphere was added for each eye, given in a bifocal lens. The media were clear; there was retinal arteriosclerosis, graded 1. There was slight benign hypertension; roentgenograms of the alveolar processes were negative. This was apparently a case of static subnormal accommodation with symptoms of presbyopia. The patient was made comfortable with bifocals.

CASE 2.—A woman, a stenographer, aged 36, had a condition that was practically identical with that of the patient in case 1. She was observed for six years, and required the addition of a +0.75 sphere, later a +1.00 sphere and finally a +1.50 sphere to relieve asthenopia for near vision.

CASE 3.—A physician, aged 33, had a low compound, hyperopic error for which he wore glasses and obtained thereby normal distant vision and an accommodative near point of from 17 to 22 cm. He was comfortable with full correction, and had no symptoms when doing close work. Although his accommodation was at a low normal for his age, it seemed to be sufficient. Symptoms, and not accommodative measurements, determine the necessity for plus additions to corrective glasses.

In group 2, which includes dynamic accommodative insufficiency, there were three cases (cases 4, 5 and 6) in which there was pupillary involvement. All were, in reality, examples of internal ophthalmoplegia, and in all the condition was unilateral. The pupils of all three of the affected eyes were dilated and remained fixed when stimulated by light. Although the patient in case 4 was unable to read fine print with the affected eye, the addition of only a + 0.50 sphere was needed for correction. In case 5, a + 1.00 sphere was required, and in case 6. a + 1.75 sphere. Apparently in none of the three cases was the accommodation completely paralyzed. The etiology in case 4 was undetermined, and only the stimulus of pilocarpine was needed to restore the action of the pupil and the ciliary muscle while the patient was under observation. The patient in case 5 had a combination of tertiary syphilis, infected teeth and tonsils. After removal of the teeth some improvement of the power of accommodation was noted, although antisyphilitic treatment was in progress. In case 6 the onset was sudden after streptococcal sore throat, and the condition had remained permanently for four years. The patient is comfortable with the unequal addition of a bifocal + 1.75 sphere for the affected eye. Two of the three patients were women, and one was a man. The ages were 34, 37 and 40, respectively. Case 4, in which the condition responded to pilocarpine, was unusual, for the use of this drug is generally regarded as of little value in the treatment for deficiency of accommodation. In this case, a few drops of a 0.25 per cent solution of physostigmine or a 0.5 per cent solution of pilocarpine sufficed to bring both the pupil and the accommodation back to normal for a few hours. Apparently this was evidence of a qualitative rather than a quantitative reduction in power.

The cases of dynamic insufficiency without pupillary involvement in group 2 are those in which I am most interested, and which furnish the main incentive for this report. Because patients who have this condition are usually less than 40 years of age, they are likely to be given correction for a static error only, unless their accommodative power is carefully investigated. I think that interpretation should be stressed, for although the accommodative near points may be taken and recorded, they are likely to be passed over as "good enough" when in reality they are not within normal range for the patient's age, and asthenopia which will not be relieved by ordinary correction may supervene. There were thirteen patients (cases 7 to 19 inclusive), four males and nine females, aged from 17 to 39 years. Only two could be said definitely to be neurotic. However, the women had a distinct asthenic tendency, which the men decidedly did not have.

In this group, the cases of outstanding interest to me were four (cases 7, 8, 9 and 10) which appeared definitely to have been caused by dental infection, and which cleared up rather promptly on the removal of infected teeth. The involvement was bilateral, but in one case it was decidedly less in one eye than in the other. Case 7 is reported in detail and corresponds in most respects with cases 8, 9 and 10.

CASE 7.—A robust man, aged 34, a bell-boy captain, complained of headaches of the migraine type which came on with close work, and which had troubled him for two weeks. Vision was 6/6. The near accommodative point was 18 cm. in each eye, which corresponds with the average accommodation for a person aged 41 years, and with the minimal accommodation for a person aged 34 years. He was not wearing glasses. There were marked insufficiency of convergence and definite conjunctival irritation. Examination performed under the influence of a cycloplegic showed that vision of the right eye was 6/6, with a +0.75 sphere, and a +0.50 cylinder of axis 88. In the left eye, vision was 6/6 with a +1.00 sphere and a +0.25 cylinder of axis 55. The media were clear and the fundus was negative. Two days after the refraction that had been made under a cycloplegic the patient accepted for the right eye a +0.25 sphere and a +0.50 cylinder of axis 75 to obtain vision of 6/7; for the left eye, a +0.50 sphere and a +0.25 cylinder of axis 65 to obtain vision of 6/7. He was given glasses of this description for constant use. After three years he returned, saying he had been comfortable with the glasses until the last month, when he had been troubled with blurring while doing near work. With glasses vision was 6/6 for each eye, and the accommodative near point for the right eye was 30 cm. while reading D 50 print; with the left eye he was unable to read D 50 print at any distance. External examination gave negative results. The pupils were normal, and marked insufficiency of convergence was evident. Manifestly he needed a +1.50 sphere added to his correction for both eyes to enable him to read D 50 print at ordinary distances. A cycloplegic was not used. The media and fundi were normal. The diagnosis was paresis or subnormal accommodation, and a general examination was made. It was reported that he had a controlled infestation with Entameba histolytica of four years' duration, which had been treated with arsenical preparations. The tonsils were small,

and not definitely infected. The Wassermann test of the blood was negative. A roentgenogram gave evidence of one definitely infected tooth. Subsequent weekly observations of the accommodative near points for one month gave the following results: right eye, 25 cm., fixed at 33 cm., 29 cm., 30 cm.; left eye, 24 cm., not able to read, 39 cm., fixed at 30 cm. The infected tooth was removed and observations of the near points were made one day, three days, three weeks and one month after operation. The results, of which the values for the right eye are given first in each instance, were as follows: 31 cm. and 30 cm., 19 cm. and 24 cm., 11 cm. and 10 cm., and 13 cm. and 12 cm. The final two values correspond with the average normal for the patient's age. After one and a half years the patient is comfortable with his original glasses, and his accommodative near points are normal for his age. This was a case of subnormal accommodative power which was dormant for several years, during which there were no symptoms. Suddenly the symptoms became very marked, apparently due to dental infection, and recovery followed promptly on the removal of an infected tooth.

CASE 8.—A woman, aged 32, a teacher, had undergone refraction, and a prescription for compound hyperopia had been given four years, and again two years, previous to the onset of the condition now to be reported. On these occasions, with the correction, her accommodative near points had been 11 cm. in the right eye and 13 cm. in the left, which were normal for her age. Subnormal accommodation was of sudden onset following prolonged use of the eyes, and the accommodative near point was at 27 cm. in one eye, and at 17 cm. in the other. After repeated confirmatory observations as to the insufficiency of accommodation, seven infected teeth were removed. After four months the accommodation had returned to a low normal. One year later it was normal. While the patient was under observation, and before the teeth had been removed, infection of a paranasal sinus depressed the accommodation even more markedly. With the resolution of the sinusitis, the condition of accommodation partially returned toward normal. Not until the dental infection was cleared up did the patient recover.

CASE 9.—A physician, aged 28, complained of difficulty in reading. Vision was 6/7. I was unable satisfactorily to determine the accommodative near point. The patient read D 50 print poorly with both eyes and without range. Examination under the influence of a cycloplegic showed the need of a +50 cylinder at an axis of 90 for both eyes. With this correction, accommodative near points were observed for three months. For the right eye they varied from 15 to 33 cm.; for the left, from 16 to 30 cm. General examination gave negative results except that one infected tooth was found roentgenographically. One month after the tooth was removed, accommodation returned to normal, 13 cm. in both eyes. Two years later it was still normal. The patient was rather asthenic.

CASE 10.—A woman, aged 29, complained of a sensation of drawing in the eyes and blurring on near vision. This was especially true of the right eye. She was distinctly neurotic. A moderate correction for compound hyperopia, with 6/6 vision and a near point of 14 cm. for both eyes, was obtained. This was the maximal accommodative near point for her age. After three years, the patient complained of increased difficulty with close work. Vision, with the correction, was 6/6 for both eyes. The accommodative near points for one month were from 22 to 25 cm. for the right eye, and from 20 to 23 cm. for the left. These correspond with the average for patients aged 43 years, and with the maximal value for patients aged 38. The general diagnosis was neurosis. Three infected teeth were removed. Six weeks later, the accommodative near points were 16 cm. in the right eye and 15 cm. in the left, which are low normal values for the patient's age. This

patient had a consistently low accommodative power for her age. This power was still further reduced by the dental infection, and returned to the previous level after dental treatment.

Cases 7, 8, 9 and 10 were of unusual interest for they seemed to form a somewhat definite clinical entity. The conditions had a definite etiology and course, and responded satisfactorily to treatment. Additional plus lenses were not required to relieve the symptoms.

The other nine cases (cases 11 to 19 inclusive) in the group of cases of dynamic insufficiency were not as definite in their course, etiology or response to treatment. In determining their cause, considerations of probability and inference had to be entertained, and treatment either was symptomatic or consisted in the use of added plus values in lenses for near work. In them the accommodative weakness was not as marked, and consequently fixation of the near point, without range, was not noted.

CASE 11.—A man, aged 38, had a temporary and marked lowering of accommodative power following appendectomy that had been performed one month before he sought refraction. The accommodative near point, with full correction afforded by +0.50 cylinders of axis 90, was 25 cm. in each eye. Since the appendectomy he had had headaches in the afternoon. Within two weeks, while he was under observation, the accommodative near point became 14 cm. in each eye, or normal, and the patient again was comfortable. General examination gave negative results except that the condition of two teeth was questionable. Apparently this case was an example of temporary disturbance of accommodation due to general asthenia after operation.

CASE 12.—A woman, aged 36, a clerk, had typical subnormal accommodation. She complained of headaches and general fatigue. She was not wearing glasses. Vision was 6/7 in both eyes. Accommodative near points were 22 cm. in the right eye and 20 cm. in the left eye, which is normal for a person aged 42 years and maximal for one aged 36. External examination and examination of the muscles gave negative results. Examination under a cycloplegic gave evidence of emmetropia. The media and fundi were normal. The patient was advised to stay under observation. Two years later she complained of blurring when doing close work. Vision of the right eye was 6/6 and of the left, 6/6-1. The accommodative near point of the right eye was 22 cm., and of the left, 21 cm., which indicated that accommodation was below normal, as it had been before. External examination and investigation of the muscles disclosed normal conditions. Manifestly, she accepted for both eyes a +1.00 sphere with vision of 6/7; the accommodative near point was 16 cm. for each eye. Examination under a cycloplegic disclosed that a +0.25 sphere and a +0.25 cylinder of axis 85 gave vision of 6/6 in the right eye; in the left, a sphere of +0.25 and a +0.25 cylinder of axis 90 gave vision of 6/7. The media were clear and the fundi normal. The prescription was as follows: right, +1.00 sphere and +0.25 cylinder of axis 85; left, +1.00 sphere and a +0.25 cylinder of axis 90 for near work. Three months later she was comfortable with glasses for near work. Vision with glasses was 6/12 for both eyes with accommodation of normal range. General examination disclosed one devitalized infected tooth which should be removed.

CASE 13.—A woman, a laboratory technician, aged 17, complained of her eyes watering and her vision blurring on close work, and of recurrent styes. Her vision was 6/7+, and her accommodative near point was 20 cm. in both eyes. That is, the accommodation was average for a patient aged 42 years, and minimal for one aged 36. She was not wearing glasses. External examination disclosed a resolving stye at the upper aspect of the inner left canthus. Under the influence of a cycloplegic and application of a +75 sphere, vision was 6/7 for both eyes. The media and fundi were normal. General examination revealed two infected teeth and diseased tonsils. The patient was advised to have the foci of infection removed and not to wear glasses. She followed this advice.

After four years the patient returned, complaining that her eyes became red after close work. Vision for the right eye was 6/6, and for the left 6/7. The accommodative near point was 19 cm. for each eye. This accommodation corresponded to the average for a person aged 41 years, and with the minimal for one aged 35. External examination and investigation of the muscles gave negative results. Under the influence of a cycloplegic, with a +0.50 sphere and a +0.25 cylinder of axis 90 for the right eye and a +0.25 sphere and +0.50 cylinder of axis 80 for the left eye, vision was 6/7 for both eyes. The media and the fundi seemed normal. When examined without a cycloplegic, the patient accepted the full correction that had been determined when a cycloplegic was used. The prescription given was as follows: right eye, +0.75 sphere with +0.25 cylinder, axis 90; left eye, +0.50 sphere with +0.25 cylinder, axis 80, for close work.

Eight months later the patient was having headaches every afternoon. Vision with correction was 6/7 for both eyes, but was blurred at the accommodative near point of 15 cm. in each eye. On external examination, slight congestion of the ocular conjunctiva was seen. Roentgenograms of the teeth were negative. Observation of the accommodative near points for four months disclosed an average of 25 cm. with correction for each eye. Finally a +1.25 sphere was added to the full cycloplegic error for use in near work. This gave a range of accommodation of from 26 to 50 cm., and a near point of 18 cm. With this the patient has been comfortable doing near work, which is very exacting. Her case is an example of persistently low accommodative power in an asthenic person, increasing over a period of five years without explanation. She was made comfortable by the addition of plus correction for near vision.

In three other cases (cases 14, 15 and 16) the condition was identical with that just described, and was relieved by the addition of plus correction for near work. One of these patients was suffering from profound neurosis; another had thyroid deficiency, and a basal metabolic rate of —23 per cent, and the third had a combination of artificial menopause with infected tonsillar tags and an infected tooth. Corrective measures were advised, the results of which are not known.

Two cases (cases 17 and 18) with the typical diagnostic ocular features of this type of case were also seen, but were not observed to completion. One of these patients gave a history of attacks of tonsillitis and scarlet fever fifteen months previous to examination.

CASE 19.—A woman, aged 38, gave a familial history of subnormal accommodation. She had attacks of blurring of near vision, which had increased in frequency. Vision of the right eye was 6/12, and of the left 6/7+. The near point for both eyes was 25 cm., which corresponded with the average accommodation

for a person aged 44 years, but was minimal for one aged 38. Slight insufficiency of convergence was noted. The patient needed, manifestly, a +1.25 sphere for both eyes, giving a vision of 6/20 and restoring near vision to the normal range. Under the influence of a cycloplegic, the error in the right eye required a —0.75 sphere and a —0.25 cylinder of axis 180 to produce vision of 6/6; the error in the left eye required a —0.50 cylinder of axis 15 to produce vision of 6/6. Residual accommodation after the cycloplegic was as follows: right eye, 1.7 diopters; left eye, 2 diopters. The media and fundi were normal. It is seen that a +2.00 sphere was added to restore near vision. General examination gave negative results, but there was a definite family history of the difficulty, in that two members of the patient's immediate family had the same ocular trouble.

There was one case (case 20) of a mixed type.

CASE 20.—A physician, aged 39, complained of headaches and blurring of vision on reading. Ten years before examination, paralysis of the ciliary muscles had followed scarlet fever. This cleared partially after tonsillectomy, but the patient had had to wear bifocal lenses off and on since then. She was wearing for the right eye a +0.75 sphere and a +0.50 cylinder of axis 50, by which she obtained vision of 6/10; for the left eye, a +0.75 sphere and a +0.75 cylinder of axis 160, by which she obtained vision of 6/10. The accommodative near points were: right eye, 39 cm., and left, 43 cm. Such accommodation would be the average for a patient aged 47 years, and minimal for a patient aged 43. Convergence was insufficient and the pupils were normal. Examination under a cycloplegic with a +1.75 sphere and a +0.25 cylinder of axis 30 gave vision of 6/7 in the right eye. A +1.75 sphere and a +0.75 cylinder of axis 150 gave vision of 6/7 in the left eye. The media and fundi were normal. Later, without a cycloplegic the patient accepted the full correction that had been determined with a cycloplegic, and was given glasses with this correction for constant use. After one month, the accommodative near points, with glasses, for both eyes were from 24 to 22 cm. which is near the lower limit of normal accommodation for her age. She was comfortable for both distant and near vision. In this case the accommodative power may have been lowered or injured by the scarlet fever and tonsillar infection of ten years before. The patient recovered sufficiently to be able to carry on, with full correction of hyperopia, although the accommodation is near the lower limits of normal for the eyes. Also, she had a history of a previous infection with Entameba histolytica that had been cured by arsenical preparations.

The cases of static or premature presbyopic accommodative insufficiency require recognition and added help for close work. The cases of dynamic accommodative insufficiency, with pupillary involvement, are really cases of unilateral ophthalmoparesis. The condition is permanent, and seems to result from some infectious insult.

Of more interest are the more fugitive cases of innervational insufficiency, without pupillary change. They are easily overlooked, and yet are associated with more intense symptoms. Their incidence seems to be less than 5 per cent among cases in which refraction is performed. In my opinion, measurement of the accommodative power and its careful interpretation are not done as a routine by many in examining patients who are aged less than 40 years. If more strict attention were

paid to this all-important phase of refraction, the incidence of subnormal states and their subsequent correction might be much higher.

Dynamic subnormal accommodation seems more likely to afflict asthenic persons, who are deficient in their reserve of nervous energy. These often have accommodative power which is persistently below the normal for their age. This already low power is still further depressed by the incidence of some intercurrent infection or debilitating condition, with resultant acute asthenopic symptoms. The condition may suddenly afflict robust persons who previously had possessed normal accommodative power, without symptoms, causing them to complain of difficulty in near vision. The onset may be sudden or gradual, and the course may be variable. Recovery may be prompt with removal of the causes, or the condition may remain permanently.

The pathology of the condition is obscure. Duane [18] expressed the belief that it is associated with lesions above the nuclei of the nerve, in the centers or tracts above these, or in their interconnecting paths, because of the lack of pupillary involvement and the fact that the condition is usually bilateral. De Schweinitz suggested that these cases might be due to a diffusible toxin of bacterial or other origin, and that these toxins might have an affinity for certain tissues. Blatt [19] suggested that there is a toxic effect on the supposed center for accommodation within the brain. Suker thought that there might be a peripheral myopathic lesion.

The symptoms are those of marked accommodative asthenopia: headaches, lacrimation, pulling and drawing sensations in the eyes, blurring of detail, conjunctival irritation and fatigue on near work. When the patients are not engaged in near work, the discomfort is much less. Insufficiency of convergence was noted in more than half of the cases; excessive convergence was present in one case.

An analysis of the etiologic factors in the thirteen cases of dynamic insufficiency without pupillary involvement disclosed systemic infection in one form or another as the one outstanding factor. In several cases more than one factor was present. In four of the cases (30 per cent) dental infection was definitely proved, and the ocular condition cleared following dental treatment. Two other cases in which dental infection was not proved were encountered. Scarlet fever, followed by tonsillar sepsis, seemed to account for two cases. Infected tonsils were also found as part of the picture in two cases. Metabolic disturbances in the form of disease of the thyroid gland were seen in one case, and artificial menopause was noted in another. Asthenia after appendectomy was noted in one case and in two cases there were histories of

19. Blatt, Nicholas: Weakness of Accommodation, Arch. Ophth. **5**:362 (March) 1931.

infestation with Entameba histolytica. Frank neurasthenia was seen in one case, and in another there was a definite familial history of low accommodative power. In two cases there was no apparent etiologic factor.

Dynamic subnormal accommodative power seems to be asthenia of the accommodative mechanism. It may be quantitative, actual marked reduction in accommodative power; it may be qualitative, inability to sustain the act of accommodation. I believe that there is a close relationship between subnormal accommodation and spasm of accommodation. In a previous paper,[20] I pointed out that spasm of accommodation is a fatigue cramp of an overworked ciliary muscle. A strained, irritated or asthenic ciliary muscle may perform in either of two ways: It may give up and suspend its function, or it may continue to act in a spasmodic fashion, weakened though it may be. Either is a manifestation of accommodative insufficiency. There is loss of that reserve, potential accommodative power that usually is held in readiness to make near vision more effective. As Blatt pointed out, there is a loss of latent accommodation, as contrasted with loss of manifest accommodation, such as is seen in paralysis.

Diagnosis of the difficulty is suggested by the presence of persistent asthenopic symptoms in younger persons, associated with near vision. If, in addition, repeated observation discloses inability to sustain accommodation at the normal values for the age, the condition is evident. In my experience, fixation of the near points at from 25 to 33 cm. from the eyes, without range, seems to be a pathognomonic feature of accommodative asthenia. In other words, the weakened accommodation seems to gather all its remaining force to carry out the act of near vision at the fixed point.

Treatment should first be directed at the correction of possible general physical causes. If this is not successful, the eyes demand added plus lenses for near work, regardless of the patient's age.

SUMMARY

Subnormal accommodation exists as a definite, clinical entity afflicting persons who have not reached the presbyopic age. It might be recognized more often if strict attention were paid to individual accommodative power and its interpretation.

Focal infection, particularly dental infection, seems to be a major cause.

Fixation of the near points, without range, is an interesting and significant feature in accommodative asthenia.

20. Prangen, A. D.: Spasm of the Accommodation, Tr. Sect. Ophth., A. M. A., 1922, p. 282.

Clinical Notes

SUBCONJUNCTIVAL HEMORRHAGE IN INTRA-OCULAR FOREIGN BODIES*

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In cases of intra-ocular foreign bodies which perforate the globe through the sclera, one would expect to find more or less hemorrhage under the conjunctiva at the site of penetration. The bleeding comes from the lacterated vessels of the conjunctiva, of the episcleral layers and of the choroid. Less frequently, when the foreign body is small and sharp and travels with great speed, perforation of the eyeball is effected without subconjunctival bleeding. In such an event the examiner is deprived of a valuable sign in the localization of the wound, and he may even experience considerable difficulty in finding it, for a small opening in the conjunctiva and episclera is quickly effaced by the reparative processes of the tissues. When a large foreign body strikes the eyeball, there is less likelihood of penetration, but there is great probability of a subconjunctival extravasation of blood from the contusion.

When the foreign body penetrates the eye by way of the cornea, the passage is through an avascular stratum, and consequently there is no subconjunctival bleeding. The foreign body flies at such a high rate of speed that penetration usually occurs with but little mechanical shock to the globe as a whole; there are, therefore, no hemorrhages under the conjunctiva such as are found in cases of contusion. The bleeding that takes place is usually within the globe, from direct injury to the vascular tissues.

I recently had the opportunity to utilize the presence of a small subconjunctival hemorrhage as an aid in the nice localization of a penetrating foreign body. A young man came to me because of an injury to the right eye sustained while he was hammering on a band of metal. Examination revealed a vertical, linear, perforating wound in the nasal portion of the cornea, well within the limbus, with a corresponding hole in the nasal sector of the iris. The presence of blood in the anterior chamber and vitreous precluded observation of the fundus. Circumstances made it difficult to obtain an x-ray photograph for purposes of localization at that time, so the patient was prepared for an immediate attempt to remove the foreign body by means of the magnet without the advantage of a film. While the patient was on the operating table, I took notice of a small hemorrhage under the conjunctiva

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* From the Herman Knapp Memorial Eye Hospital.

nasally, not far from the caruncle. During the preliminary examination, I had paid scant attention to this hemorrhage. The gravity of the injury had completely overshadowed the insignificant ecchymosis, but on reflection I was unable to account for the presence of blood nasally except on the basis that the foreign body had reperforated the eyeball at that point. Accordingly, a dissection of the conjunctiva was made in the region of the hemorrhage, and without difficulty a piece of steel was removed from beneath the conjunctiva outside of the sclera. Such an instance of double perforation is rare, to be sure, but the possibility should not be overlooked in cases of intra-ocular foreign bodies where there is bleeding under the conjunctiva which is not readily explained.

Ophthalmologic Review

PAPILLEDEMA *

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Papilledema has long been of particular interest to ophthalmologists, and the literature in regard to this subject is large. The present review, however, will be limited to those contributions that are particularly concerned with the method of formation and the pathology of papilledema.

The theories in regard to the formation of papilledema may be divided into the mechanical and the nonmechanical types. The mechanical types may be concerned with an interference with either the lymph flow or the vascular supply of the optic nerve. The nonmechanical types may be due either to an inflammation or to a toxic agent.

At the present time one or another of the mechanical theories holds the preference. It is of historical interest that the first theory advanced, namely, that of Graefe,[1] in 1860, was mechanical in its view. Graefe believed that the tumor of the brain pressed either directly or indirectly on the cavernous sinus and produced edema of the nerve, venous engorgement and retinal hemorrhages because of the venous stasis produced in the ophthalmic vein. Shortly afterward he abandoned this opinion, when Sesemann [2] demonstrated the free anastomosis that exists between the ophthalmic and the facial veins. Recently, the rôle of the intracranial venous sinuses as an etiologic agent has been revived by Swift.[3]

The observations of Swift are based on the observation of three cases, two in adults, each of whom had an angle tumor, and one in a newborn infant, who died in status epilepticus on the morning of the third day after birth. In each of the adults autopsy showed the right transverse sinus to be fully developed while the left was only rudimentary. In the first case, in which the tumor occurred on the side of the rudimentary sinus, there were no signs of increased intracranial pressure and no papilledema developed. In the second adult, the tumor developed on the side of the normal sinus, and in this case there was bilateral papilledema and other symptoms of increased intracranial pressure, such as

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* From the Department of Ophthalmology of the University of Pennsylvania.

1. Graefe: Arch. f. Ophth. **7**:58, 1860.

2. Sesemann: Arch. f. Anat. u. Physik., 1869, p. 154.

3. Swift, G. W.: The Transverse Sinus and Its Relation to Choked Disk, Arch. Ophth. **3**:47 (Jan.) 1930.

nausea, vomiting and headache. Autopsy of the child showed that the trabeculae, which usually disappear in the transverse sinus, were still present and that the jugular foramina were both rudimentary in character and size. In his summary he stated that "occlusion of the jugular bulb does not cause choked disk in a normally symmetrical development of the sinus, but a marked choked disk, unilateral at first and then bilateral whenever there is an absence of the left transverse sinus or a marked decrease in its caliber."

Because of the lesser importance that is at present assigned to the nonmechanical theories, it may be well to review these first.

THE NONMECHANICAL THEORIES

As early as 1879, Gowers [4] formulated an inflammatory theory, and in 1881, Leber [5] believed the cause to be the development of a toxic material by the intracranial disease. This material, he believed, passed into the cerebrospinal fluid and then into the nerve, nerve sheath and papilla. Inflammation was set up with edema and infiltration as secondary manifestations. This inflammatory theory was supported on experimental grounds by Deutschmann,[6] and on anatomic grounds by Lawford and Edmunds,[7] Elschnig [8] and Scimeni.[6]

A forward extension of an edematous process originating in the brain was believed to be the cause by Sourdille [9] and Kampherstein.[10]

Sourdille believed that the fluid was forced from the third ventricle over the chiasm and then into the optic nerves; these nerves became swollen and were strangulated in the foramina with a resulting venous and lymph stasis of the papilla and the optic nerve. Kampherstein believed that the edema of the brain extended into the optic nerve, through the lamina cribrosa into the papilla, and produced an incarceration of the intra-ocular end of the nerve.

A disturbance in the vasomotor system has been assumed as the cause of the swelling of the disk by various authors. Benedict [11] thought that this swelling was brought about by pathologic changes in the sympathetic nervous system. Loring [12] and Hughlings Jackson [13]

4. Gowers, W. R.: A Manual and Atlas of Medical Ophthalmoscopy, ed. 4, London, J. & A. Churchill, 1904, p. 74.

5. Leber: Tr. First Internat. M. Cong., London **3**:53, 1881.

6. Cited by Parsons: Pathology of the Eye, London, Hodder & Stoughton, 1904.

7. Lawford and Edmunds: Tr. Ophth. Soc. U. Kingdom **3**:138, 1883.

8. Elschnig: Arch. f. Ophth. **41**:179, 1895.

9. Sourdille: Arch. d'opht. **21**:378, 1901.

10. Kampherstein: Klin. Monatsbl. f. Augenh. **42**:501, 1904.

11. Benedict: Allg. Wien. med. Ztschr. **13**:21, 1868.

12. Loring: New York M. J. **35**:561, 1882.

13. Jackson, Hughlings: Roy. London Ophth. Hosp. Rep. **4**:389, 1863.

thought that there was a compression of the nerves that controlled the circulation and nutrition of the optic nerve. Adamkiewicz [14] regarded the condition as a neuroparalytic inflammation.

There has been no experimental or pathologic support of the vasomotor theories until recently. In 1919, Kornder [15] was able to produce papilledema in six of a series of ten dogs in from three to ten days by the injection of 1 cc. of melted paraffin through a trephine opening in such a way as to block the aqueduct of Sylvius. The first sign that he noted of increased intracranial pressure was a prominence of the retinal veins. In another series of experiments he was able to produce a swelling of 3 diopters in one eye and of 2 diopters in the other in forty minutes in a dog by the subdural injection of fluid. He found that if the inhibitory action of the vagus was removed by atropinization of the dog previous to the carrying out of the procedure mentioned, no papilledema was produced, although the highly increased intracranial pressure was continued for as long as three and a half hours. He did not believe that the increased venous pressure shown by an engorgement of the retinal veins was due to a local cause such as direct pressure on the nerve structure or the central vessels, but that it was simply a local manifestation of a general high venous pressure which was present in the venous system of the whole body. As a result of the high venous pressure in the retinal veins, there occurs an excessive transudate of lymph into the disk and retina, with the production of the clinical picture of papilledema. In regard to a vasomotor etiology, he makes the following statement, "whether this high venous pressure is entirely due to the accentuated inhibitory action of the vagus consequent to central stimulation from the high cerebrospinal fluid pressure may be a question open for further investigation."

THE MECHANICAL THEORIES

The theories of the mechanical type have as their starting point the anatomic demonstration by Schwalbe [16] of the connection between the subarachnoid spaces of the brain and the optic nerve.

Schmidt [17] injected a solution of Berlin blue under pressure into the subdural space of rabbits and dogs. He found that the nerve sheath was distended, but that the outer layer was not colored. Neither was the tissue of the optic nerve itself colored, but there was a heavy precipitate and staining with blue at the point of junction with the bulb. The color

14. Adamkiewicz: Ztschr. f. klin. Med. **28**:28, 1895.

15. Kornder, L. H.: Hydrocephalus and Choked Disk in Dogs, Arch. Int. Med. **23**:197 (Feb.) 1919.

16. Schwalbe: Arch. f. mickr. Anat. **6**:1, 1870.

17. Schmidt: Arch. f. Ophth. **15**:193, 1869.

did not extend into the sclera or into Tenon's space. By increasing the pressure under which the injection was made, Schmidt was able to demonstrate a complete filling of the lamina cribrosa with the fluid and dye. He concluded that the lamina cribrosa contained a canal system that is in direct communication with the subarachnoid space. He believed that papilledema was due to this edema, by the fluid forced in at the lamina cribrosa, and that this in turn caused an incarceration of the intra-ocular end of the optic nerve. Schmidt-Rimpler [18] also believed that there was a direct driving forward of the cerebrospinal fluid through the lamina cribrosa, with an edematous infiltration of the nerve head as a direct result. According to this theory, the venous congestion was secondary.

Manz [19] was not able to demonstrate, in the manner noted, a system of lymph canals in the lamina cribrosa that communicated with the subarachnoid space. Since the nerve sheath was distended by the fluid under pressure, he formulated an explanation of papilledema based on compression of the central vessels of the retina as they passed through the lamina cribrosa. There resulted an edematous swelling and infiltration of the nerve head.

Bordley and Cushing [20] have produced experimental work that supported the views of Manz and Schmidt-Rimpler. Fluid was introduced into the subdural space under pressure. When the fluid pressure was in excess of the venous pressure, a venous engorgement appeared in the retinal veins and a measurable amount of swelling of the papilla was produced. If the pressure was made nearly equal to the arterial pressure, a high grade of venous engorgement resulted with a high swelling of the disk. In other experiments, the nerve sheath was ligated distal to the chiasm with sufficient pressure to cause a venous obstruction without interfering with the arterial circulation. In these experiments only a venous engorgement occurred and no swelling was apparent. From these experiments they did not believe that venous stasis alone was capable of producing papilledema.

Cushing and Bordley [21] also recorded another experiment in which a globe and optic nerve were isolated in a dog, a constricting band was placed about the optic nerve close to the optic foramen, and pressure was made just short of shutting off the central artery of the retina. This resulted in a venous stasis but no swelling or edema. They concluded

18. Schmidt-Rimpler: Arch. f. Augenh. **18**:152, 1888.

19. Manz: Deutsches Arch. f. klin. Med. **9**:339, 1872; Klin. Monatsbl. f. Augenh. **3**:281, 1865.

20. Bordley, James, Jr., and Cushing, Harvey: Observations on Choked Disc, J. A. M. A. **52**:353 (Jan. 30) 1909.

21. Cushing, Harvey; and Bordley, James, Jr.: Bull. Johns Hopkins Hosp. **20**:95, 1909.

from this that "simple stasis in the retinal veins alone fails to produce anything more than the condition of venous engorgement which accompanies choked disk and never leads to a definite edema of the papilla."

Blocking the lymphatic drainage of the optic nerve has been regarded as the primary factor in the production of papilledema by Rochon-Duvigneaud [22] and by Liebricht.[23] This idea has been developed and modified in the several contributions of Schieck.[24]

In a microscopic examination of the eyes in four cases, three of which showed recent papilledema and one of which showed postpapillitic atrophy, Schieck found no evidence of inflammation. He found that the perivascular lymphatic spaces were open and that there was marked distention of the perivascular spaces about the central vessels. These findings he considered characteristic. When a gelatin mixture was injected into the intervaginal space, he was able to demonstrate the material in the tissues about the central vessels. He believed that papilledema was caused by the entrance of cerebrospinal fluid under increased pressure in the preformed perivascular lymph spaces of the axial bundle of the optic nerve and along the central vessels into the disk. In a more recent contribution, he has demonstrated the connection between the perivascular lymph spaces and the intervaginal space of the optic nerve by injecting a mixture of prussian blue and oil into the optic nerve sheath in a freshly enucleated eye.

On the basis of animal experiments and the injection of fluid into the subdural and subarachnoid spaces, Levinsohn [25] opposed the view of Schieck, that cerebrospinal fluid was forced forward, but advanced the opinion that the swelling of the disk is due to a checking of the outflow of the lymph from the vitreous cavity. He also believed incorrect the views of Deyl [26] and Knape,[27] to be referred to later, in regard to the compression of the central vessels of the retina in the dural sheath.

The view of Berens, Smith and Cornwall [28] corresponds closely to that of Levinsohn. They stated "that experiments on injections in animals conducted under the direction of Dr. J. E. Sweet, as well as those reported by Gifford and Wegefarth have convinced us that there

22. Rochon-Duvigneaud: Arch. d'opht. **25**:401, 1895.

23. Liebricht: Arch. f. Augenh. **91**:84, 1922.

24. Schieck: Arch. f. Ophth. **113**:157, 1924; Deutsche med. Wchnschr. **39**:10, 1913.

25. Levinsohn: Arch. f. Ophth. **81**:15, 1912.

26. Deyl: Wien. klin. Rundschau **13**:165, 1899.

27. Knape, E.: Mitt. a. d. Augenh. d. Carolinsch Med. Chru. Inst. zu Stockholm **10**:1, 1909.

28. Berens, Conrad; Smith, H. T., and Cornwall, L. H.: Changes in the Fundus and in the Blood Pressure in the Retinal Arteries in Increased Intracranial Pressure, Arch. Neurol. & Psychiat. **20**:1151 (Dec.) 1928.

is a lymphatic drainage from the eye ball along the central vessels, and it is our belief that blockage of this drainage is an important factor in the production of the edema."

The theories advanced by Saenger and by Behr have to do with pressure on the optic nerve where it traverses the optic foramen. Saenger [29] believed that, although there was an initial distention of the neural sheath with cerebrospinal fluid, as the intracranial tension rose there was a compression of the optic nerve, and the communication of its sheath with the intracranial cavity was cut off by the pressure of the frontal lobe from above and the temporal lobe from the outer side on the dural fold that surrounded the nerve at its place of entry in the bony canal. In this way there was an interruption of the lymph flow with a consequent papilledema. The untenability of this mechanism is obvious since the publication by Foster Kennedy [30] of his series of cases of tumors of the frontal lobe in which pressure on the optic nerve led to a retrobulbar neuritis with the formation of a central scotoma and primary optic atrophy on the side of the lesion with a papilledema on the opposite side. Gowers and Horsley had previously pointed out this same syndrome.

While Behr [31] adopted the view that there is a compression of the nerve at the opening of the bony canal, he believed that in addition the lymph stasis of the sheath compresses the central vein in the nerve with a production of venous congestion and engorgement of the vessels of the fundus. The clinical observations of concentric contraction of the fields with a sparing of the papillomacular bundle are the supporting facts of this theory of compression at the bony canal. In his publication of his observations on the examination of forty-five cases of papilledema in serial section, Behr noted particularly a peripheral area of nerve fiber degeneration, marked proliferative changes in the endothelium of the vessels and intervaginal spaces and furrowing of the optic nerve at the point of entry into the bony canal. He also stated that he had never observed a compression of the central vein in the intervaginal portion. This does not correspond to the findings of other authors to be referred to later. In experiments on dogs, Behr found that when the optic nerve was constricted from 0.5 to 1.5 cm. behind the bulb, an edematous swelling of the papilla is developed in from two to sixteen hours after operation, and that this swelling is similar to that of papilledema in man. Paton and Holmes, who do not accept pressure on the optic nerve as the cause of papilledema, believe that it is important in cutting short its course and the production of secondary atrophy.

29. Saenger: Deutsche Ztschr. f. Nervenh. **41**:99, 1911.
30. Kennedy, Foster: Am. J. M. Sc. **142**:355, 1911.
31. Behr, C.: Neurol. Centralbl. **30**:66, 1911.

Lillie, in a discussion of the paper by Swift, stated that in his opinion Behr's theory better than any other solves the clinical problems that arise in connection with papilledema.

Compression of the central vein of the retina has been advanced as the primary fact in the production of choked disk by various authors, who, however, disagree about the point at which this compression takes place.

Deyl, who made serial sections of the optic nerve in a case of papilledema, found distention of the intervaginal space with acute bending and compression of the vein as it enters the dura. Knape also described a compression of the vein by the edematous swelling of the dura. He did not believe that this was a constant finding because of a disappearance of the edema and pressure after death.

The observations of Dupuy-Dutemps,[32] who later believed that the compression of the central vessels took place in the intervaginal space, were first made from the examination of one each of the eyes in three cases of tumor of the brain. In one of the cases the optic nerve had become atrophic. From these examinations he thought that venous compression was of primary importance and that it took place at the point where the vein traversed the dural membrane. This statement was not made on the direct observation of the compressed vessel, but on the observation of an edema of the optic nerve, which rapidly diminished at the place of exit of the central vessels. In later observations, his view was that the venous compression was within the subarachnoid space of the optic nerve.

Merz [33] performed a series of experiments on rabbits, in which he produced choked disk by the subdural injection of a saline solution. The optic nerves were sectioned later. He found the principal cause to be a compression of the central vessels as they passed through the intervaginal space.

Paton and Holmes [34] have based their opinion on a detailed histologic study of sixty eyes. They believe that the primary factor is a local increase in the venous pressure in the central vein. They stated that, "in our opinion, therefore, the edema of the papilla that constitutes tumor papilledema is, in the first place, due to the venous engorgement that results from the rise of intravenous pressure that is necessary in order that circulation should be maintained in the intervaginal portion of the vein where this is subjected to an increased sheath pressure. The increased sheath pressure is also the origin of the second factor, the obstruction to the lymph drainage from the papilla." They found that

32. Dupuy-Dutemps: Thèse, Paris, 1900.
33. Merz: Arch. f. Augenh. **41**:325, 1900.
34. Paton and Holmes: Brain **30**:389, 1911.

the lumen of the central vein was still generally well open until it passed through the pia and was lying in the subarachnoid space, when its walls were collapsed and its lumen was reduced to a slit. Although sixty eyes were examined, the optic nerve was cut in serial section in but five cases. They did not state in how many of these five cases the slitlike lumen of the vein was encountered.

Foster Moore,[35] from a review of the various theories of papilledema, believed that compression of the central vein in the subarachnoid space was the one most acceptable.

MISCELLANEOUS THEORIES

Several contributions have been made that merit notice, although they do not follow directly in the classification of theories noted.

Tilney [36] pointed out the existence of a small canal extending out from the third ventricle above the optic nerve and chiasm. This tubular extension persists in adult animals only in its proximal portion and extends along the nerve for 0.75 mm. Tilney believed that this was of significance in the production of papilledema in which there was an accumulation and retention of fluid in the ventricles of the forebrain. Kornder made serial sections through the region of the optic chiasm in several animals with moderately distended brains, but found nothing definite.

Parker,[37] in experiments on dogs and monkeys, did an Elliot trephine on one eye in each animal. After the trephine opening had healed, the intracranial pressure was increased, and he found that the nerve in the eye with the lower tension, the trephined eye, was the first to show papilledema. When papilledema appeared in each eye, the eye with the lower tension showed the higher swelling. Meyer [38] had previously recorded clinical evidence that pointed to the same fact. He recorded two cases; a swelling of 4 diopters was present in one, and in the other a swelling of 1.5 diopters was observed in the hypotonic eye, while the eye with normal tension showed no swelling.

The clinical observation that the papilledema frequently first appears on the side on which the brain is involved has been confirmed experimentally by Rados.[39] He produced symptoms of increased intracranial pressure in rats by inoculation with brain sarcomas and was able to

35. Moore, Foster: Medical Ophthalmoscopy, Philadelphia, P. Blakiston's Son & Company, 1925, p. 22.

36. Tilney: Anat. Rec. **10**:250, 1916.

37. Parker, W. R.: The Relation of Choked Disk to the Tension of the Eyeball, J. A. M. A. **67**:1053 (Oct. 7) 1916; Tr. Am. Acad. Ophth., 1924, p. 77.

38. Meyer: Klin. Monatsbl. f. Augenh. **51**:373, 1913.

39. Rados: Arch. f. Augenh. **79**:199, 1910.

produce ophthalmoscopic and histologic changes in the disk that resembled papilledema in man.

The pathologic changes in papilledema have been studied by Kampherstein and Elschnig, are reviewed in Parsons'[40] "Pathology of the Eye" and are presented in detail by Paton and Holmes. Much of the following description follows the observations of Parsons and of Paton and Holmes.

Sections of marked cases of recent papilledema show a mushroom type of outline of the disk, with a widely distended sheath. Kampherstein and Elschnig found this distention of the sheath in about 65 per cent of their cases, although in only from 35 to 40 per cent of the cases was this distention extreme, and it appeared to be least in the earliest cases. The subarachnoid space is the one chiefly involved, the arachnoid membrane usually being closely applied to the dural sheath. The subarachnoid space is frequently filled with a coagulated exudate and in the more chronic cases with an endothelial proliferation, but the latter, in the opinion of Paton and Holmes, is never sufficient to cause compression of the nerve. The distention of the sheath is usually most marked several millimeters behind the globe, and this has led to the designation of this type of distention as ampuliform.

Particular attention has been focused on the changes within the disk, and here the general opinion is that the primary change is an edema. This edema, which first makes its appearance in the anterior layers of the lamina cribrosa, causes a wide separation of the nerve fibers of the disk, and extends in the nerve fiber layer for as much as a disk diameter into the retina. Although the nerve fiber layer is chiefly affected, there is also some edema of the internuclear layer and of the internal nuclear layer.

The retina adjacent to the disk is displaced laterally and sometimes with it the pigment layer, which may be thrown into a series of elevations. The normal uniform curve of the fibers is changed so that the more laterally placed fibers take an S-shaped course. Paton and Holmes found that in severe cases the internal limiting membrane near the disk was raised in small elevations. These contained an exudate and were traversed by stretched or ruptured fibers of Muller. They were seen more frequently on the macular side of the disk.

Edema of the intra-orbital part of the nerve was noted by Kampherstein in 60 per cent of his cases. He found the edema to be characteristically interfascicular in location. Paton and Holmes noted in this regard that there is a definite difference between the vessel-bearing portion of the nerve and that part behind the entrance of the central vessels. They found that the edema rapidly disappeared behind this point. This

40. Parsons: Pathology of the Eye, London, Hodder & Stoughton, 1904.

difference had been noted previously by Dupuy-Dutemps and was one of the chief facts that led him to the formulation of his theory. A subpial edema, often quite marked, was emphasized by Paton and Holmes. On the other hand, Ulrich stated that subpial edema was seldom found.

In the denser posterior layers of the lamina cribrosa, no alteration in position or separation of the fibers has been noted. The anterior layers of the lamina, corresponding to the pars choroidea, are carried forward to form an arc convex anteriorly, and the fibers are so stretched and separated that this layer is recognized with difficulty. Attention was first called to this feature by Schweigger.[6] Elschnig, who found this feature in 91 per cent of his cases, thought it the most constant anatomic difference between papillitis and papilledema. Kampherstein found it in 79 per cent of his cases and Liebricht in all of twelve cases.

In a few cases only one side of the disk appears swollen. Paton and Holmes explained this as due to an especially dense band of connective tissue in the lamina cribrosa extending from the central vessels toward the outer edge of the disk.

Most observers have found that the inflammatory changes in cases of recent papilledema are very scant. There may be a slight small round cell infiltration in the connective tissue about the central vessels. There may also be a similar slight infiltration in the loose connective tissue about the central vessels and at the edge of the nerve where the vessels make their exit. There is a wide dilatation of the vessels of the swollen papilla, with hemorrhages, both along the vessel sheaths and just beneath the internal limiting membrane. Edema about the central vessels in the nerve stem has been a rather constant finding.

The degenerative changes have been divided by Paton and Holmes into a primary and a secondary type. The primary type was seen most marked near the surface of the swelling and in the lateral bulging of the nerve fibers. By ordinary stains these were seen as opaque homogeneous areas. They described in the disk "cystoid bodies" similar to those found in the retina in albuminuric retinitis, in vascular lesions and in the region of local injuries. These were described as "varicose degeneration of the nerve fibers." After a certain stage of development, they undergo fatty degeneration, so that they were not seen in the latter stages of papilledema. The secondary degeneration was seen as a crescentic area on the edge of the nerve. These areas have been studied and importance has been given to them by Behr.

In the later stages of papilledema, there is a proliferation of neuroglial tissue, with a gradual shrinkage of the tissue of the swollen disk.

News and Notes

EDITED BY DR. JOHN HERBERT WAITE

GENERAL NEWS

Massachusetts Eye and Ear Infirmary Alumni Meeting.—The Alumni Association of the Massachusetts Eye and Ear Infirmary held its annual meeting at Boston on October 30 and 31. Clinics and papers on eye, ear, nose and throat subjects were given by members of the consulting, visiting and resident staff during the two days, followed by the annual dinner on the evening of the second day.

Ophthalmologic Exhibit.—Application for space for an ophthalmologic exhibit at the next session of the American Medical Association should be filed by those interested no later than Jan. 10, 1932, with Dr. Parker Heath, 1025 David Whitney Building, Detroit.

The John Markle Eye Clinic.—The Seamen's Church Institute of New York announces the opening of a second clinic for serving merchant seamen. The first clinic was opened in July, 1931, to render dental service. The second clinic will be known as the John Markle Eye Clinic, after the donor, and will be open each Tuesday and Friday morning to merchant seamen of every nationality who are not beneficiaries of other agencies. Dr. Conrad Berens and Dr. G. Paccione will be in charge of the clinic.

Colby College.—Colby College has instituted a complete physical examination of all new students to be done each fall by the Thayer Hospital staff. For the purpose of a check-up, the examination will be repeated the senior year. The ophthalmologic examination includes an external examination (a history of symptoms of asthenopia, tests for vision and for muscle balance with the Maddox rod) and an examination of the fundi, without dilation of the pupil. This year the entire male student body, 450 men, received the examination.

Axenfeld Memorial.—A suitable memorial to Theodor Axenfeld is to be erected in the University Eye Clinic in Freiburg. The many who have received instruction and drawn inspiration from this indefatigable, courteous scientist, will be glad of the opportunity of subscribing to the fund. Donations are to be sent to the Universitäts Augenklinik, Tübingen, Germany, with the designation "Axenfeld-Erinnerungsspende."

Ophthalmic Examination.—The American Board for Ophthalmic Examinations will hold an examination in New Orleans on Monday, May 9, 1932, at the time of the meeting of the American Medical Association. Applications for this examination should be procured from the secretary, Dr. William H. Wilder, 122 South Michigan Avenue, Chicago, and should be sent to him at least sixty days before the date of the examination.

SOCIETY NEWS

Paris Ophthalmological Society.—The Paris Ophthalmological Society held its annual meeting on Nov. 15, 1931, at 12 Rue de Seine. The program was as follows: 10 a. m. Professor Marquez, Skiascopy with Cylinders; M. Polack, Practical Demonstration with Dalton's Color Tests, and M. Leopold Levi, Endocrine Glands with Reference to the Pathology and Therapeutics of the Eyes. 2:45 p. m. Discussion of the report of M. Cerise and M. Robert Thirel, Pathologic Anesthesia of the Cornea.

DEATHS

We regret to announce the death of Dr. George Strong Derby, occurring Saturday, December 12. A full obituary will appear in a latter issue of the ARCHIVES.

CORRECTION

In the paper by Dr. Benjamin Friedman entitled "The Development of the Lens: Its Significance in the Interpretation of Lenticular Abnormalities," which appeared in the October issue (ARCH. OPHTH. **6**: 558, 1931), the following errors occurred:

On page 561, seventh line from the bottom, "surface ectoderm by the lens of the stalk" should read, "surface ectoderm by a stalk."

On page 568, lines 1 and 12, and page 569, line 1, "optic vesicle" should read "lens vesicle."

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Color Sense

CONGENITAL TOTAL COLOR BLINDNESS. W. E. FRY, Am. J. Ophth. 13: 1064 (Dec.) 1930.

The author discusses the explanation of congenital total color blindness according to the different theories of color vision. The case reported is the eighth in this country. A woman, aged 23, came under observation because of day blindness. Vision was so poor on a bright day as to prevent her getting about, though she could do so very well in dim light. She was totally color blind. Corrected vision was 6/60 in the right eye and 6/30 in the left. The eyes were normal externally, though a short lateral nystagmoid movement could be detected on ophthalmoscopic examination. The fundi and the fields were normal.

W. S. REESE.

Congenital Anomalies

CONGENITAL ANGIONEUROTIC EDEMA WITH GERMINAL DYSPLASIA AND MASKED HYPERTHYROIDISM COMBINED WITH EDEMA OF THE LID. D. MICHAIL, Ztschr. f. Augenh. 73: 337 (March) 1931.

The author relates a case in which there was a definite history of germinal dysplasia consisting of high hypermetropia, pupillary membrane and partial iris coloboma. In addition, the vascular system of the left side of the face and lid showed a definite pathologic alteration. There was also evidence of endocrine disturbance, for the patient showed signs of infantilism at puberty. Michail describes symptoms that were manifestations of irritability of the vagus nerve and also active hyperthyroidism. The edema developed in the spring and lasted until autumn. He believes that the edema was produced by the sun's ultraviolet rays on the area of congenital malformation of the mesoblastic tissues of the patient's face.

A. M. YUDKIN.

Experimental Pathology

EXPERIMENTAL HYPHOMYCETES INFECTIONS OF THE EYES. W. JADASSOHN and K. REHSTEINER, Klin. Wchnschr. 10: 308 (Feb. 14) 1931.

The work of Bloch on experimental fungus infections of the skin showed that the fungi grew in the horny layer of the surface epithelium and also in the ectodermal portion of the skin. Just how these microorganisms would act in the ectodermal portions of the eye, especially the cornea and lens, seemed of importance. In six guinea-pigs the

corneas were scarified and cultures of Achorion quinckeanum were introduced. Slit-lamp control over a period of three months showed absolutely negative results. Injections of the following fungi were made into the lenses of guinea-pigs and rabbits: Achorion quinckeanum, Achorion schoenlienii, Trichophyton gypseum and Microsporon audouini and furfur. Positive slit-lamp and microscopic results were obtained for all organisms except Microsporon audouini and furfur. The area of growth in the lens, as noted by the slit-lamp, was most interesting. Only in the deep, "dead" inner cortical nucleus was there any growth. The authors believe that this demonstrates the fact that such organisms as Hyphomycetes have a predilection for dead or poorly nourished tissues. That the lens is not a homogenous body but is divided into zones, as shown by Vogt, seems to have been demonstrated. L. L. MAYER.

SPIROCHETAL CONTENT OF THE EYES AFTER SUBSCROTAL VACCINATION WITH SYPHILIS. K. KAMADA, Klin. Wchnschr. **10:** 1116 (June 13) 1931.

Igersheimer found that 10 per cent of rabbits' eyes in experimental syphilis showed metastatic keratitis. The infection occurred less often in other localizations in the eye. Nichols and Hough found choroiditis and Reasoner bilateral cataract with gumma of the ciliary body, iris and lens. Browne and Pearce found iritis. The author used the subscrotal method of Nichols. Five rabbits' eyes examined between the thirty-seventh and the two hundred and thirty-eighth day following vaccination showed positive findings in the cornea, while the lens, vitreous and aqueous were negative. The author believes that these results are important because spirochetes are not usually found in the organs of experimental animals These findings contradict those of Uhlenhuth, which were negative even in brain tissues. A bibliography accompanies the article. L. L. MAYER.

General

OPHTHALMOLOGIC PANORAMA. W. GILBERT, Med. Klin. **27:** 929 (June 19) 1931.

A résumé of modern advances in ophthalmology is given, under the following headings: (1) operations on the eye, pathology and therapy, (2) diseases of the brain and of the eye, (3) refraction and accommodation.

In the first division brief reference is made to Birch-Hirschfeld's technic for cataract operation, Elschnig's work on intracapsular cataract extraction in conical cornea and Luntz' experimental investigation of the Gonin operation. The author also considers Brambergen's paper concerning cataract after thyroidectomy, Pillat's investigation of keratomalacia. Poos' pharmacologic study of the alkaloids and, finally, the effects of roentgen treatment on the retina as presented by Hoffmann. In the second division, the neurologic aspect of lesions of the brain and optic nerve is commented on. The works of Igersheimer on chiasmal tumors, of Rochat concerning Lindau's disease, of Hirsch on pituitary tumors and of Goldstein and Wexler on neurofibromas are criticized.

The third division outlines first the work of Marchesani on Levinsohn's myopia in monkeys, Seka's investigation of the school myopia contracted in Russian and Turkish children, and Roggenkämper's report of transitory myopia following injury to the eye. Then follows a discussion of Blatt's work on changes of the pupil and accommodation in lead poisoning, Zur Nedden's on detachment after accidents in highly myopic persons, Hessberg's on one-eyedness, Weekers and Hubin's on stereopic vision in single-eyed persons as it relates to their ability to drive a motor vehicle and Coppez' on the necessary physical aptitude of the automobile driver. A complete bibliography of the works discussed follows the article.

L. L. MAYER.

General Diseases

ACTINOMYCOSIS OF THE VISUAL ORGAN. O. THIES, Klin. Monatsbl. f. Augenh. **86**: 55 (Jan.) 1931.

A woman, aged 28, had been suffering from epiphora of the right eye for over a year; purulent discharge had developed during recent months. Local treatment and extirpation of the tear sac failed to cure the condition. The area of the lower canaliculus was slightly prominent and congested. Slitting of the lower canaliculus revealed a white, friable mass, the size of a millet seed, which, under the microscope, proved to be actinomyces. The irritation of the conjunctiva disappeared soon after the operation, and there was no recurrence.

The right eye of the second patient, a laborer, had been inflamed for several weeks. A small, gray vesicular prominence in the cornea resembled the beginning of a keratocele; its surroundings were grayish. Otherwise the eye presented no pathologic changes. The efflorescence disappeared soon, but recurred about three months later. At this time the eye was free from irritation, except at the temporal portion of the limbus. A yellowish-white spot was seen at the site of the prominence described, and a gray zone adjoining it, toward the pupil. Formations of actinomyces could be recognized by use of the slit-lamp, and microscopic examination confirmed the diagnosis. The corneal defect healed within a few days after thorough curettage. The invasion of the actinomyces could have happened only through a slight injury. Thermocautery may have to be resorted to in some cases.

K. L. STOLL.

AN UNUSUAL OCULAR DISTURBANCE IN THYROID DISEASE. H. GASTEIGER, Wien. klin. Wchnschr. **44**: 887 (July 3) 1931.

This case is reported because a perusal of the literature fails to reveal findings of a like nature. It concerns a man, 47 years old, who had typical symptoms and signs of toxic thyroidism. In addition, a slight swelling of the lower lids was noted on first examination. Following conservative treatment, which was without avail, a partial thyroidectomy was done. The general condition was benefited by operation; nevertheless, the swelling of the lids increased and also affected the upper lids. The skin over the swelling was firm, and there was no pitting on pressure. Because of an increasing bilateral exophthalmos, it was necessary to

cover both eyes with a watch glass shield, as the weight of the lower lids contributed to the failure of closure of the eyes. After treatment with diuretic preparations, thyroxine, etc., the swelling gradually decreased leaving the lower lids hanging like deflated bags over the nasolabial folds. The exophthalmos decreased and vision was enhanced. The author feels that the condition was probably due to a postoperative myxedema, which agrees with Stower's theory. Two excellent photographs and a bibliography accompany the article. L. L. MAYER.

Lacrimal Apparatus

PURULENT DACRYO-CYSTITIS TREATED WITH IODIZED SESAME OIL. A. LOEWENSTEIN, Klin. Monatsbl. f. Augenh. **85:** 811 (Dec.) 1930.

Iodized sesame oil 40 per cent has been used in ophthalmology for about thirty years. The author uses a thin preparation, manufactured by Merck, for treatment in purulent dacryocystitis. It has yielded prompt results, especially in old people. In some cases the purulent discharge, and even the epiphora, disappeared after one or two irrigations of the tear sac. The disadvantage of this method is found in the necessity of repeating it as often as it may be required. It is justified, in the author's opinion, because the shock caused by chiseling and trephining in various operations can be avoided frequently. Toti's, West's and other operations, of course, retain their place. Irritation has never been observed following the application of a 10 per cent solution of the iodized oil. K. L. STOLL.

Lens

POSTERIOR LENTICONUS. R. I. LLOYD, Am. J. Ophth. **13:** 968 (Nov.) 1930.

The author discusses the embryology of posterior lenticonus and related ocular anomalies. He considers hyaloid traction as the cause of posterior lenticonus. Two cases are reported. An extensive bibliography is appended. W. S. REESE.

CALCIUM DETERMINATIONS ON CATARACTOUS HUMAN LENSES. P. W. SALIT, Am. J. Ophth. **13:** 1072 (Dec.) 1930.

The author comments on the relatively large amount of calcium in cataractous lenses and its absence in normal lenses although the fluids around the lens contain appreciable amounts. Fifty-one cataractous lenses were examined, but no correlation between the calcium values and the duration of the cataract was found. The following summary is given: Analyses of forty-five human cataractous lenses for calcium gave an average value of 35.4 mg. per hundred grams, with a range of variation of from 2.8 to 108 mg. No calcium could be demonstrated in five lenses with incipient cataract. One black cataract, the only one examined for calcium, likewise contained none. W. S. REESE.

CONCERNING LAMELLAR MEMBRANES OF THE ANTERIOR SURFACE OF THE LENS. T. B. HOLLOWAY and A. COWAN, Am. J. Ophth. **14:** 189 (March) 1931.

The authors review the literature on zonular lamella as to its origin, embryology and extent. In case 1, that of a man, aged 64, the biomicroscope revealed a lacelike appearance due to broad areas of fuzz or scalelike opacities in the midzone of the lens. These were attached to the capsule, and many had upturned margins. The expanse of the lesions was greatest toward the periphery. Shortly afterward, this patient developed pain and increased tension. Similar cases in the literature are discussed and their relation to glaucoma stressed. Case 2 was that of a man, aged 70, who had worked in a rolling mill for forty years as a puddler and heater. With the biomicroscope, the curled margins of a homogeneous, transparent membrane could be noted in the pupillary area of the lens capsule. These margins were slightly serrated and glistening, and projected into the anterior chamber where a few small tags floated freely. In case 3, a laborer, aged 60, exhibited a thin, homogeneous, transparent membrane on the anterior surface of the lens, as seen with the biomicroscope. The membrane was curled upward and rolled like a scroll. The lens capsule below this membrane was perfectly smooth and transparent. Tension of this eye was 33.25 (Schiötz). According to the authors, most of the patients in the foregoing cases have been exposed to heat for a prolonged time. They discuss this phase, quoting Elschnig, Kraupa, Vogt and others, and its relation to glassblowers' cataract.

In case 4, the patient, aged 11, had had a linear extraction and presented a penetrating scar near the limbus at 11 o'clock. Behind the plane of the iris there was a thin, transparent, structureless membrane that stretched over the pupillary area and was attached to the corneal scar. This type of membrane has been attributed to the vitreous. This and another type seen following intracapsular extraction are to be referred to in a subsequent communication.

W. S. REESE.

THE PRESENT STATUS OF THE INDICATIONS FOR CATARACT OPERATION. K. SAFAR, Wien. klin. Wchnschr. **44:** 324 (March 6) 1931.

The general condition of the patient and the local condition of the eye must be investigated. Age is no contraindication. Diabetes is treated by dietary methods and the use of insulin. High blood pressure should be reduced to prevent bleeding. The coughing or sneezing of bronchitic or asthmatic patients should be well controlled. Foci of infection in the tonsils or teeth and suppurations of the skin should be eliminated. Infections of the tear sacs and conjunctiva must be brought to an asceptic status to prevent infection of the eye following operation. The time for operation is decisively influenced by the degree of cloudiness of the lens. The stages are described under the headings of incipient, intumescent, mature and hypermature cataract. The cataract does not necessarily have to be "ripe," although it is safer, but modern methods are used to cause artificial ripening or, on the other hand, an intracapsular operation may be done. Even though the two eyes may not be useful together when only one is operated on, due

to the difference in the size of the image, the gain in visual field to the side makes it well worth while. As long as practical vision is present there is no need to rush into an operation, as the clouding often takes many years to become a definite hindrance to vision. The advantages and disadvantages of operative procedures are discussed relative to intracapsular or extracapsular methods. The fact that glaucoma may occur in a cataractous eye, due to swelling of the lens, must always be kept in mind. It is important to test for perception and projection and to determine the prognosis for vision. Determinations of the visual fields and blind spot will help to indicate any defect of the retina or optic tract in order to make a guarded prognosis. If the intra-ocular tension is raised, due to the cataract, it is rational to perform an extraction.

L. L. MAYER.

Neurology

EYE CHANGES OBSERVED IN PARETIC PATIENTS AFTER TREATMENT WITH MALARIA. C. P. CLARK, Am. J. Ophth. **13**: 946 (Nov.) 1930.

The author remarks the few ophthalmologic observations on malarial treatment for syphilis. He discusses the optic nerve involvement in relation to malarial therapy and gives a brief history of the origin of this therapy. The mode of action of this therapy is discussed under the following headings: (1) elevated body temperature, (2) leukocytosis, (3) increased body metabolism with elimination of toxic substances, (4) antibody formation and (5) vasodilatation.

Intravenous injection of 2.5 to 4 cc. of blood drawn from the donor was given at once to the recipient. The malarial paroxysms began from five to seven days later. The treatment is terminated by quinine, depending on the physical condition of the patient. The author gives the statistical material of fifty cases studied with reference to (1) central visual acuity, (2) motor anomalies, (3) pupillary behavior, (4) eyeground changes and (5) visual field changes. Improvement was noted in the eyegrounds of twelve patients and in the visual field in four cases. Twenty-two patients with bilateral and six with unilateral Argyll Robertson pupils recovered normal reactions. The following conclusion is made: The improvement that takes place in dementia paralytica after malarial fever therapy must be attributed to stimulation of the defense reaction of the reticulo-endothelial system, elevated body temperature and hyperemia. The benefit to the ocular tissues and visual pathways must be produced in this manner. It is suggested by these observations that malarial inoculation therapy is beneficial for syphilitic involvement of the optic nerve and visual pathways. It is to be hoped that other observers will note and publish the eye changes that occur after malarial treatment for syphilitic disease of the central nervous system.

W. S. REESE.

SUPRASELLAR (INTRACRANIAL) AND ORBITAL MENINGIOMAS. R. ARGAÑARAZ, Arch. de oftal. de Buenos Aires **6**: 3 (Jan.-Feb.) 1931.

Recent progress in the localization of intracranial tumors, in the surgical procedures for their removal and in their anatomic characteris-

tics have greatly modified ideas in regard to them. The author refers to meningiomas, first described by Cushing, which may develop in the meninges, the optic nerve sheath or its intracranial coverings. In regard to the latter he points to the distinguishing feature of an early development of the visual symptoms with later development of those indicating pituitary involvement, the reverse occurring in tumors affecting primarily Rathke's pouch. The author considers the different sites along the course of the optic nerve where these meningiomas may develop, with their course and peculiar symptoms. The visual symptoms are at first limited to a single eye and are first established by a study of the visual fields. The changes in the fields vary according to the site of the compression. Bitemporal hemianopia is exceptional in suprasellar meningiomas. The author does not find anything characteristic in the color fields such as mentioned by Cushing. Orbital meningiomas are more apt to develop in children than in adults; the suprasellar variety is usually found later, between the ages of 40 and 50, rarely before 30 or after 60. The early symptoms are visual; next, those of endocrine origin, and last, those arising from an increase of intracranial pressure. The author reports in detail six clinical cases.

C. E. FINLAY.

IS THERE A SYMPTOM COMPLEX INVOLVING THE IRIS IN TABES AND PROGRESSIVE PARALYSIS? P. VANCEA, Ztschr. f. Augenh. **73**: 254 (Feb.). 1931.

Tiere describes a triad of symptoms characterized by atrophy of the iris, irregular contour of the pupils and anisocoria in progressive paralysis. Many authors on the continent believe that the Argyll Robertson phenomenon is produced by atrophy of the iris. In order to ascertain whether the foregoing assumptions are correct, Vancea examined twenty-two patients with progressive paralysis and six with tabes dorsalis. Observations were made with the slit-lamp, corneal microscope and the differential pupilloscope of Hess. As a result of this examination, eight cases of atrophy of the iris, four of irregular contours of the pupils, thirteen of anisocoria, two of atrophy of the iris accompanied by anisocoria and one of atrophy of the iris and irregular contour of the pupil were found. Vancea concludes that there is no definite symptom complex between the iris and tabes dorsalis and progressive paralysis.

A. M. YUDKIN.

Physiology

FURTHER CONTRIBUTION TO THE KNOWLEDGE OF POSTTRAUMATIC TRANSFERENCE OF STIMULATION WHICH WAS OBSERVED AS CONSENSUAL INTRA-OCULAR HYPERTONY. SVEN LARSSON, Acta Ophth. **9**: 85, 1931.

An 8 year old child was to be operated on for alternating internal strabismus, but a slight amount of 1 per cent procaine hydrochloride and epinephrine was injected accidently into the vitreous chamber, and immediately the cornea became hazy and the tension of the eyeball was lowered. The composition of the normal intra-ocular fluid was altered, the optic disk became blurred and the venous system was abnormally

engorged. On the following day, the same condition prevailed in the injured eye, and a similar but milder change appeared in the eye on which operation was not performed. This eye returned to normal in three weeks, whereas it took the damaged eye seven weeks to return to its normal status. The author reports this case to show that there is probably some truth in the theory that it is possible to transfer stimulation from the injured eye to that of the normal eye and that it is manifested by a lowering of the intra-ocular pressure. This is a supplement to the article appearing in *Acta ophthalmologica* (8: 261, 1930).

A. M. YUDKIN.

Retina and Optic Nerve

HEMORRHAGE INTO THE OPTIC NERVE SHEATH. A. FAVORY, Arch. d'opht. **48:** 81 (Feb.) 1931.

Favory first points out that although anatomic observations indicate the rarity of this condition, the possibility of such a diagnosis must not be forgotten, and furthermore it should be thought of every time sudden visual disturbances occur in the course of a meningeal hemorrhage whether or not fundus changes are present. He discusses the ophthalmoscopic signs under four headings. The first consists of the rapid appearance of retinal change about the disk extending toward the macula, which develops within a few hours. The second heading deals with the modification of the blood vessels, constriction of the arteries and dilatation of the veins. The third describes the hemorrhages which sometimes spread into the vitreous. The variations in the occurrence of choked disk are discussed under the fourth heading. In some instances, no ophthalmoscopic changes are to be seen, and the subjective symptoms then form the basis of a diagnosis. Loss of vision comes on suddenly and may or may not be complete. In those cases in which it is not total, field changes consisting in peripheral contraction will be found, the amount of loss depending on the damage to the nerve. Pupillary symptoms parallel visual loss.

Hemorrhage into the optic nerve sheath is never primary, and according to Gonin almost always has an extra-orbital cause. Autopsy evidence shows that they may be due to injury or meningeal hemorrhage accompanying alcoholism, diabetes or albuminuria. They are very rarely due to fracture of the optic canal.

The author describes two types of cases, one traumatic, the second a hemorrhagic meningitis. The pathology is discussed. The author disagrees with Paton as to the mechanism of the production of choked disk. He does not think that interference with the nerve is so much a factor as the presence of intracranial hemorrhage acting as a tumor. He cites a third case in support of this contention. He presents the theories suggested by various authors to explain the retinal hemorrhages. He himself believes them to be dependent on the pressure on the central retinal vein in its vaginal path.

The pupillary changes are easily explained when vision is completely lost. When some vision is retained, the pupillary phenomena are less easily explained. Two cases of Terrien's are cited.

S. B. MARLOW.

AN UNUSUAL CASE OF BURN OF THE MACULA CAUSED BY GAZING INTO THE SUN. GRUETER, Klin. Monatsbl. f. Augenh. **86:** 104 (Jan.) 1931.

A man, aged 28, gazed into the sun for several minutes during a schizophrenic attack. He noticed a decided decrease of his vision, especially in his left eye, soon after the psychic excitation had ceased. Examination, a month later, showed the right eye to be normal, with the exception of a yellowish spot, surrounded by a red area, in the macula. A sharply defined white scar with an angular outline was seen in the macula of the left eye. The vision was reduced to 6/12 in the right eye and to 6/60 eccentric, in the left. A disk-shaped central scotoma of from 2 to 3 degrees diameter was found in the right field of vision, and a disk-shaped, absolute scotoma of 2 degrees, with a relative scotoma for colors of from 3 to 4 degrees in the left.

K. L. STOLL.

TRAUMATIC DETACHMENT OF THE RETINA. HAITZ, Klin. Monatsbl. f. Augenh. **86:** 106 (Jan.) 1931.

The author reported the case of a healthy man, aged 49, who fell on his back and suffered a sector-shaped detachment of the retina downward. The rupture was located at the ora serrata. The eye had been normal when examined fifteen months prior to the accident. The patient had lost his other eye, due to an iron splinter, twenty-two years before. Immediately after the fall, he noticed a single flash in his eye, and two months later the vision began to fail. The mechanism of the detachment, in the author's opinion, was that the vitreous became jerked backward and torn off its base. Portions of the ciliary epithelium, according to Salzmann, becoming detached simultaneously, deprive the margin of the retina of its attachment. Aqueous humor of the posterior chamber and vitreous fluid succeeded in lifting off the retina. This traumatic detachment changed but slightly in the course of three years, with preservation of 5/36 vision. Gonin's operation, therefore, had not been resorted to. Three similar cases were reported during the discussion.

K. L. STOLL.

IGNIPUNCTURE IN DETACHMENT OF THE RETINA. R. DEUTSCHMANN, Ztschr. f. Augenh. **73:** 127 (Jan.) 1931.

From an experience of over thirty years with detachment of the retina, Deutschmann does not subscribe to the theory advocated by Gonin, Vogt and others. On the contrary, he believes that the tears and holes frequently found accompanying this condition are symptoms of a disturbance in the subretinal tissue. He hypothecates that the hole must come secondary to a detachment in the summit of the "blister" which is produced by subretinal fluid under increased pressure. He points out that even Gonin, Vogt and others conceded the fact that the openings of the holes and tears point toward the vitreous cavity rather than to the choroid, and hence strengthen his assumption.

He quite agrees with Vogt that ignipuncture performs the task of closing up the tears and holes, but reattachment takes place only when the retina flattens itself out. The procedure allows a marked subretinal

and preretinal emptying of the fluid and produces an abundant transudation from the ciliary body into the vitreous cavity. Should the latter fail, the chance of a good result is often lost. Not all detachments should be subjected to ignipuncture, even though apparently fresh, until the case has been thoroughly studied and its etiology understood, for the retina and choroid frequently shrink following this operation. The author finds it necessary, at times, to cut the tissue at the ora serrata to allow further give in the retina and choroid, so as to avoid further tears.

A. M. YUDKIN.

Trachoma

OPERATIVE TREATMENT OF TRACHOMATOUS PANNUS BY IMPLANTATION OF BUCCAL MUCOSA (DENIG'S PROCEDURE). V. VEJDOVSKY, Rev. internat. du trachome **8**: 57 (April) 1931.

A report of fifteen eyes operated on by this method and observed for fourteen months is made. No case of relapse of the pannus was noted. The operation is done as described by Denig, with Loewenstein's modification of the peritomy. The sutures are similarly used as in cataract extraction done at the Clinic Quinze-Vingts. A good bibliography is appended.

L. L. MAYER.

THE TREATMENT FOR TRACHOMA WITH COLD SOLID CARBON DIOXIDE. HENRIK SJÖGREN, Acta ophth. **8**: 296, 1930.

A critical review of the treatment for trachoma by cold applications is given and the results obtained commented on. Not all investigators are in accord with the efficacy of this treatment. The following procedure is recommended by Sjögren to obtain favorable cures. The eye is anesthetized with cocaine, and procaine hydrochloride-epinephrine is injected into the upper border of the tarsus, so that the whole culdesac is made visible. It is not necessary to make an injection into the lower tarsal area. A carbon dioxide pencil is employed to freeze the tissue. It is made by passing the carbon dioxide gas into a leather bag, and the condensed ice which is formed is then packed into a small round metallic tube. The solid pencil is then applied to small areas on the tissue for an interval of from fifteen to twenty seconds until the whole diseased conjunctiva is frozen. An even pressure of approximately 1.5 to 2 Kg. should be applied to the tissue with the pencil when freezing. An excavation of about 4 mm. is necessary for satisfactory results. Care must be taken to cover the prongs of the forceps with rubber tissue so that they do not come in contact with the frozen area. Bleeding is frequently produced when this precaution is not taken. The tissue changes to a porcelain color, becomes hard and excavated when properly frozen. In order to avoid discomfort for the patient and damage to the cornea, it is advisable to wait until the tissue assumes a reddish hue before turning the lid over into position. For the next few days the tissue is edematous, and frequently a pseudomembrane is formed. The upper layer exfoliates, and the deeper strata gradually heal. In the same manner, the lower lid, plica and caruncle are treated.

The culdesac is washed daily with mercuric oxycyanide (1:2,000 or 1:4,000) until the irritation disappears, and then copper acetate ointment or copper sulphate stick is applied.

The foregoing procedure may be repeated every week or ten days. It is best to wait, however, for the reaction to clear up before the next treatment is given. Patients with acute cases of trachoma should not be subjected to this ordeal until some caustic application has been previously tried. The author reports a number of cases in which this procedure was used.

A. M. YUDKIN.

Uvea

ENDOGENOUS INFECTIONS IN THE ETIOLOGY OF ACUTE IRITIS. T. G. SCHNABEL, Am. J. Ophth. **14:** 223 (March) 1931.

The author presents ten tables in which he analyzes the causes and various other pertinent conditions in twenty cases of iritis. He gives the following summary: 1. Many inflammatory attacks in the uveal tract subside promptly after elimination of endogenous infections by appropriate measures. 2. Patients with inflammatory lesions of the uvea or of a portion of this tract have generally but not invariably one focally infected site, and more frequently two or more such foci. The incidence of focal infection in monocular cases may be as great and greater, as is shown in the control group cited. 3. The most frequent foci in our group and in other groups similarly studied are to be found in tonsils, teeth, accessory sinuses, prostates and the respiratory tract. The gastrointestinal tract and the female pelvis must very rarely be the seat of a primary infection leading to a secondary eye lesion. 4. Some patients with ocular inflammation seem to have no possibilities for endogenous infection. 5. Some iritic attacks subside without focal infection therapy. 6. Some patients have recurrent attacks. These may be due to overlooked foci, inadequate therapy, or the development of new foci. 7. Acute and chronic iritis and iridocyclitis have been produced experimentally by a technic or technics that seem to show with reasonable certainty that focal infection is an etiologic possibility for inflammation of the iris. 8. Other conditions may be necessary to make a focus of infection serve for ocular metastasis. Such conditions may be an anemia, tuberculosis, syphilis, diabetes, lack of local tissue immunity or some electivity on the part of the invading organisms. Poor local drainage in the infected focus may change it to one acting as a portal of entry. 9. In our series tuberculosis may have been an etiologic factor of greater importance than would appear on first inspection, if the intracutaneous tuberculin reaction is a reliable test. 10. Many causes at one time assigned as responsible for iritis do not appear in this group. 11. It appears that the cause of acute inflammation of the uveal tract, including iritis, is not always easily discovered. In all probability a variable number of conditions and circumstances must coexist to bring about such eye lesions. Such variables apply to both the host and the invading microorganisms. At the present time it seems that endogenous infections play a large part in the production of acute iritis and that their elimination is a therapeutic desideratum.

W. S. REESE.

THE ETIOLOGY OF THE KRUKENBERG SPINDLE. A. BAUER, Ztschr. f. Augenh. **73**: 360 (March) 1931.

An analysis is made of thirty-two cases of pigmentary deposits on the posterior layer of the cornea. The distribution is known as the Krukenberg spindle. Bauer finds that the condition is associated with myopia, is found in adults over 25 years of age and seems to be more prevalent in women. The lesion differs from that process ordinarily associated with persistent pupillary membrane. The Krukenberg spindle is different from other pigment deposits on the posterior aspect of the cornea. The pigment has an endogenous origin and is a product of the pigment areas of the eye. Destructive processes in the pigment cell transmit in a slow and chronic manner the catabolic substances into the intraocular fluid, and then the pigment is deposited on the posterior layer of the cornea because of the presence of a constant convection current, known as the Türk-Ehrlich phenomena, in the anterior chamber of the eye. The spindle-shaped deposit of pigment is usually vertical and occupies an area on the posterior endothelium of the cornea opposite the pupil. Bauer believes that the downward movement of the stream and an alteration of the endothelial cells of the cornea are responsible for this formation. Myopia and megalocornea seem to favor this deposition of pigment. The Krukenberg spindle is not a congenital process.

A. M. YUDKIN.

Visual Tracts and Fields

THE INFLUENCE OF PREGNANCY ON THE VISUAL FIELD. J. P. JOHNS, Am. J. Ophth. **13**: 956 (Nov.) 1930.

The author reviews the literature and the conflicting reports. The present study comprised an attempt to ascertain any change in the endocrine status and standardized technic for examinations of the visual fields both during pregnancy and for a reasonable period post partum. Patients with venereal disease were excluded. Studies in vital function were made on admission and after delivery, and in seventeen cases stereoscopic roentgenograms were made of the sella turcica in the eighth or ninth month of pregnancy and again a number of weeks post partum. The twenty-nine patients were of good intelligence, so that the eye examinations were quite satisfactory. Form fields were measured at monthly intervals until the beginning of the ninth month, when they were made weekly. After delivery and return from the hospital, form and color fields were measured and additional form fields taken at monthly intervals for at least three months. The Feree-Rand perimeter was used.

Five tables are given, and the following summary and conclusions made: 1. The investigation showed definite concentric contraction of form and color fields in the majority of the cases. 2. The blind spot showed enlargement in the majority of the cases. 3. Enlargement of the blind spot and concentric contraction of the form and color fields are consistent with a general reduction of retinal vitality, in that the parts of the field involved in the contraction are those having a minimum retinal sensitivity. The endocrine studies show a lowered vitality through the pregnant state, tending to return to normal post partum. This parallels the visual field studies in this series of cases. 4. These visual field studies

do not show the characteristics of pathologic changes of the pituitary gland associated with enlargement great enough to cause pressure, such as bitemporal hemianopia, superior temporal cutting, scotomas in the cecocentral or paracentral area, or interlacing of color fields. These measurements are apparently not of sufficient magnitude to have arisen from either pressure or contact. 5. On the basis of the visual fields, the roentgenographic measurements, and the investigations of vital functions, it seems warrantable to conclude that the field changes in pregnancy probably depend on functional modification rather than on enlargement or vascular changes in the pituitary gland. Whether these field changes are to be regarded as physiologic or pathologic is a question only to be decided by further study.

W. S. REESE.

Vitreous

TRAUMATIC HERNIA OF THE VITREOUS INTO THE ANTERIOR CHAMBER. HAITZ, Klin. Monatsbl. f. Augenh. **86:** 105 (Jan.) 1931.

A genuine hernia of the vitreous into the anterior chamber was found after the patient's eye had been struck by a shot from an air rifle. The hernia could be recognized with the slit-lamp by a distinct reflex cone. The prolapse, similar to others observed, possessed great stability. It could not be observed with oblique illumination. It is essential for any prospective removal of the traumatic cataract to establish its presence. Its origin is explained by preservation of the anterior limiting layer of the vitreous during the impact, allowing a portion of the vitreous to prolapse in toto into the anterior chamber.

K. L. STOLL.

Society Transactions

EDITED BY DR. GEORGE S. DERBY

AMERICAN OPHTHALMOLOGICAL SOCIETY

Sixty-Seventh Annual Meeting, June 1-3, 1931, Asheville, N. C.

ARNOLD KNAPP, M.D., *Chairman*

EXOPHTHALMOS IN INFANTILE SCURVY. DR. J. H. DUNNINGTON, New York.

This article appeared in full in the ARCHIVES, October, 1931, p. 731.

DISCUSSION

DR. DORLAND SMITH, Bridgeport, Conn.: Dr. Smith reported a similar case in an 11 months old baby, fed for three months exclusively on Mellin's food and water. Hemorrhage, edema of the upper lids and proptosis were extreme. Without any treatment other than changing to a milk diet, the entire process was arrested and cleared up in three or four days.

TRIBROMETHANOL AS AN ANESTHETIC IN OPHTHALMIC SURGERY. DR. F. A. DAVIS, Madison, Wis.

The author records his experiences with tribromethanol in ninety ophthalmic operations. The advantages claimed for the method are the freedom from preoperative anxiety and from the complications met with in children, non-English speaking or nervous patients when operated on under local anesthesia. After a colonic flushing, 0.1 Gm. of the drug per kilogram of body weight is given by rectum, thirty minutes before operation. In most cases the anesthesia was perfect. In only a few was it necessary to administer a small amount of ether. The eyes remained motionless during operation, requiring double fixation for the cataract incision. Seventy-five per cent of patients wakened quietly, and most patients showed complete amnesia with respect to the operation. Only two patients showed symptoms of asphyxia, which were due to improper care by the anesthetist. This may be avoided by maintaining free respiration, which may require a breathing tube. Only fifteen patients vomited after operation, and in these cases the vomiting was slight and not a cause of complications. There were no deaths. A remarkable reduction of intra-ocular tension was noted in all cases, which was of value in operations for glaucoma.

DISCUSSION

DR. ARNOLD KNAPP, New York: Dr. Knapp asked how long the lowered intra-ocular pressure endured, and whether it was followed by a sudden rise.

Dr. E. V. L. Brown, Chicago: Dr. Brown spoke of the routine use of morphine-scopolamine as an adjuvant to local anesthesia in the conjunctival sac in all cases in which administration of morphine-scopolamine the night before does not induce nausea and vomiting.

Dr. John W. Burke, Washington, D. C.: Dr. Burke asked whether wrinkling of the cornea as a consequence of reduced ocular pressure occurs following the use of tribromethanol.

Dr. F. A. Davis: Dr. Davis closed the discussion, stating that he did not know how long the ocular pressure remained subnormal, and that he had not observed anything more than the striate keratitis that sometimes follows extraction.

Unilateral Traumatic Ophthalmoplegia Externa and Interna. Dr. Dunbar Roy, Atlanta, Ga.

Dr. Roy outlined the case of a man who struck his head on a sharp object beneath the water while swimming. The left eye became immediately blind. A wound through the lower lid was repaired. Twenty-four hours later there was left optic atrophy, complete ptosis and ophthalmoplegia. There was no ecchymosis or proptosis. Treatment by diathermy and sodium iodide internally was followed by improvement. Six months later, the ophthalmoplegia and ptosis had disappeared entirely. No vision was recovered, but the left pupil reacted to light.

Congenital Absence of Lacrimal Puncta and of Canaliculi. Dr. Everett L. Goar, Houston, Texas.

This article appeared in full in the Archives, October, 1931, p. 724.

DISCUSSION

Dr. P. C. Jameson, Brooklyn: Dr. Jameson suggested the insertion of a tongue of the conjunctival mucous membrane through the incision into the sac itself, and suturing it there, with a view to getting epithelization.

Dr. William C. Finnoff, Denver: Dr. Finnoff stated that he had seen Dr. Goar's case, and that a good functional result had been obtained.

Dr. Arnold Knapp, New York: Dr. Knapp asked whether any external deformity was present, and how large the new duct was.

Dr. Everett L. Goar: Dr. Goar replied that a no. 8 sound drops readily into the canal, and that the only deformity is a little scar at the margin of the lower lid.

It might be well to implant a conjunctival tongue in the way Kraupa does his operation, but it is a formidable procedure.

The Cataract of Postoperative Tetany, with Report of Three Cases. Dr. C. S. O'Brien, Iowa City.

This article appeared in full in the Archives, October, 1931, p. 724.

DISCUSSION

Dr. William H. Wilder, Chicago: Dr. Wilder cited the case of a woman of 35 years in whom bilateral cataracts were found two years

after thyroidectomy followed by tetany. Each lens showed a peculiar incipient cataract made up of radiating lines of tiny vacuoles, and other vacuoles were scattered through the deeper layers of the cortex. Dr. Wilder had no biomicroscope at that time and had to rely on the high power loupe and the ophthalmoscope. Tests of the blood, blood pressure, urine, etc., gave negative results. Tests for the amount of calcium in the blood or for calcium metabolism were not made. Tetany was probably induced because of defective action of the parathyroids, or possible injury to them at the time of operation. This case may therefore be classed as one following tetany with probably defective calcium content in the blood.

Dr. F. Phinizy Calhoun, Atlanta, Ga.: Dr. Calhoun was interested in the observation that spontaneous postoperative hemorrhages may be a complication, unless the proper treatment is given with calcium lactate, a high calcium diet and parathyroid extract before and after operation. He reported the case of a woman, aged 29, who, six weeks after thyroidectomy developed convulsions, and six months after that noted failing vision. Examination of the eyes showed that throughout the anterior and posterior cortex of each lens, surrounding a large irregular nucleus, were myriads of dots and exclamation-point-like opacities radiating toward the center. Four years after thyroidectomy, a preliminary iridectomy was done on the left eye. A mild hemorrhage into the anterior chamber remained for six days. Three months later, the lens was extracted. A severe hemorrhage into the anterior chamber occurred on the fifth day. It gradually grew less, but fresh blood was detected for two months. During this time the patient menstruated three times in forty-six days.

In spite of continuous daily treatment consisting of 60 grains (39 Gm.) of calcium lactate, 5 grains of parathyroid extract (0.32 Gm.) and 40 units of parathyroid extract-Collip, the blood calcium was 6.7 mg. per hundred cubic centimeters of blood, slightly below normal. A persistent increase in intra-ocular tension, uncontrolled by miotics or epinephrine, made a trephine necessary. One year later the vision and tension were normal.

Dr. Edward C. Ellett, Memphis: Dr. Ellett reported a somewhat similar case.

Dr. Francis H. Adler, Philadelphia: Two years ago Dr. Freeman, of the department of physiology of the University of Pennsylvania, performed a complete parathyroidectomy on three dogs for Dr. Adler. They were then watched for three or four months. Unfortunately, no studies were made of their blood serum. Dr. Adler emphasized the point that in spite of the fact that the dogs had complete parathyroidectomy, they were kept on a high calcium intake and parathyroid gland, and they did not develop tetany. Two of the dogs began to show changes after one month, and Dr. Adler confirmed Dr. O'Brien's observation that the changes are subcapsular and confined to the anteroposterior cortex in the region of the capsule. So far as could be observed, after four months, the changes were just these fine punctate dots. There was no way of judging whether they interfered with vision. At the end of that time the dogs were in good health, and no other changes were noted.

DR. JONAS S. FRIEDENWALD, Baltimore: Dr. Friedenwald reported another unusual ocular complication following thyroidectomy. The patient was a woman about 40 on whom thyroidectomy had been performed, and as a result not only symptoms of parathyroid insufficiency but hypothyroidism developed. About two months after operation she began to complain of visual impairment, and on examination at that time was found to have beginning cataracts of the subcapsular variety, and in addition definite neuroretinitis with central scotoma. Dr. Friedenwald went through the literature at that time and was able to find only five cases of retrobulbar neuritis or neuroretinitis associated with either hypothyroidism or parathyroid tetany. Of course, in this woman's case it was impossible to decide which of the two dyscrasies was the cause of the neuroretinitis.

DR. FREDERICK A. DAVIS, Madison, Wis.: Dr. Davis cited the case of a patient who developed failing vision three months after the thyroid operation. She had convulsions, however, which lasted for two years. A preliminary iridectomy was performed in one eye, and a hemorrhage developed at the time of the first dressing three days after operation. This eye was lost due to destructive inflammation. Of course, at first it was thought that there was simply hemorrhage due to lowered calcium content. The coagulation at that time was normal, three minutes. This patient at the same time had a tonsil tag and a 2 plus Wassermann reaction.

Operation was performed a year later by a colleague in another city, who did a successful combined extraction in the fellow eye.

DR. SANFORD R. GIFFORD, Chicago: Dr. Goldmann thyroidectomized a number of dogs and was able to keep them alive for a period of from six to eight months, and in one case for over a year. He found that the opacities developed only during the periods of tetany. When the dogs were controlled so that they did not have tetany, opacities did not develop, and when they were free for several weeks or a month he was able to watch the clear lens material being laid down over the opacity; then he would let them have tetany, and more opacities would develop, and so on. His picture shows one dog with at least six rings of opacity in the lens, separated by clear lens material. This, of course, explains why opacities in one case may be described as being subcapsular, and in another as deeper in the lens.

DR. C. A. CLAPP, Baltimore: Dr. Clapp added a case history, that of a woman 50 years of age who developed posttetany cataract, and who also developed intra-ocular hemorrhage seven days after extraction, even in spite of treatment with calcium prior to operation.

DR. C. S. O'BRIEN, Iowa City: Of course, it is necessary for calcium to be maintained in order to keep the lens protein in normal condition, and it may be that a lowered amount of calcium has something to do with it—that the electrochemical changes mean the production of a lowered amount of calcium. On the other hand, it does not fit every case, because those patients whose spinal fluid had been tested were found to have normal amounts of phosphorus and calcium. The third patient continued to develop cataracts in the absence of any attacks of tetany. Since calcium acts on the coagulability of the blood, whether the hemorrhages could be due to lowered coagulation time is another ques-

tion. So far as could be determined, the coagulation time was normal in all three cases.

CONCERNING CYCLODIALYSIS IN SIMPLE GLAUCOMA. DR. HARRY GRADLE, Chicago.

The author believes Heine's operation has not been employed as generally as it should be. He advises cyclodialysis only in cases of simple glaucoma without any signs of inflammation, cases that have shown failure of vision and fields under the use of miotics, especially when the tension can be kept down almost, but not quite, to normal by miotics. It is advised especially in cases in which the field is cut down near to the fixation point, as here a loss of field following operation is much less likely to occur than after iris-cutting operations. Gradle reports on twenty-seven cases answering to the foregoing requirements, which had been followed for one to five years after operation.

A special indication is after an iridectomy that has failed to control the tension. Of the twenty-seven cases, the two failures were in cases not properly chosen for cyclodialysis, as the condition was too far advanced. In four cases partial success was obtained, but other operations were necessary later. In twenty-one cases, vision, tension and fields have remained satisfactory. At least 80 per cent of successes may be expected if the indications are closely followed. Gradle considers the operation considerably less liable to postoperative complications than other glaucoma operations, and devoid of the danger of late infection.

DISCUSSION

DR. T. B. HOLLOWAY, Philadelphia: Dr. Holloway stated that he has used cyclodialysis for the simple glaucoma, for marked contraction of the field where iridectomy is practically prohibitive and after failure in maintaining reduction of tension following iridectomy or trephining. Cyclodialysis should be followed by massage of the globe beginning twenty-four hours after operation and continued daily.

DR. W. H. WILMER, Baltimore: Cyclodialysis followed by massage and miotics has been of value in chronic simple glaucoma in elderly people with marked vascular hypertension, rather a dangerous situation in which to do a scleral trephine. In those cases in which in addition to increased intra-ocular tension there is a mild uveitis, results nearly always have been failures.

DR. GEORGE E. DE SCHWEINITZ, Philadelphia: Dr. de Schweinitz reported a favorable personal experience with cyclodialysis. After cyclodialysis, miotics act better than prior to its performance, and cyclodialysis seems to be the safest kind of operation in elderly patients.

DR. WILLIAM ZENTMAYER, Philadelphia: Dr. Zentmayer added his testimony to the value of cyclodialysis in the class of case that Dr. Gradle mentioned. He has thus saved several eyes with glaucoma secondary to obstruction of the central vein of the retina.

DR. M. J. SCHOENBERG, New York: The fact that the field of vision is limited need not deter one from doing a Lagrange or a trephine operation. In my experience I have seen quite a number of cases in which the trephine operation was done, and the limited field of vision did not decrease.

DR. ARNOLD KNAPP, New York: Success depends on little traumatization. Also, results after cyclodialysis are to a great extent negative, whenever hemorrhage occurs during or after operation.

DR. HARRY S. GRADLE, Chicago: Dr. Gradle did not agree with the chairman that hemorrhage into the anterior chamber nullifies the results. Homatropine should be used at the conclusion of the operation, and usually for two or three days after the operation. Miotics are seldom used until a week or ten days after operation, until all inflammatory reaction has subsided, and then in rather weak concentration and at comparatively infrequent intervals, say once a day.

INTRASCLERAL NERVE LOOPS. DR. A. B. REESE, New York.

This article appeared in full in the ARCHIVES, October, 1931, p. 698.

DISCUSSION

DR. T. B. HOLLOWAY, Philadelphia: Dr. Holloway spoke of a case which showed characteristic markings and also a malignant growth, cataract and glaucoma.

THE CIRCULATION OF THE AQUEOUS. DR. JONAS FRIEDENWALD and DR. H. F. PIERCE, Baltimore.

This article will appear in full in a later issue of the ARCHIVES.

DISCUSSION

DR. A. MAGITOT, Paris, France: Dr. Magitot asked why the enucleated eye maintains a tension of 10 mm. of mercury.

DR. PETER C. KRONFELD, Chicago: Dr. Kronfeld has measured the production of aqueous after blocking the normal outlets of the anterior chamber with paraffin oil, and his figures agree fairly well with those of Dr. Friedenwald.

DR. ARTHUR M. YUDKIN, New Haven, Conn.: The introduction of a foreign metallic body into the eye alters the character and the rate of production of the aqueous humor. If some method other than needle puncture could be employed so that the irritation of the eye could be controlled, we would have an ideal way of determining whether there is a constant flow from the aqueous chamber, and also how much flows within a given time.

DR. F. H. VERHOEFF, Boston: The origin of the fluid in the eye appears to be from the ciliary process, its chief outflow through the canal of Schlemm, and the canal of Schlemm acts as a safety valve for any increase in pressure. When you enucleate an eye you naturally lower the pressure because you have taken the blood pressure out of the uveal coat and that makes the pressure lower in the whole eye. You have a diminished outflow, you have no venous flow through the canal of Schlemm, and it makes a difference in the flow of fluid out of the eye.

DR. JONAS S. FRIEDENWALD, Baltimore: The introduction of a needle into the eye is a rather serious insult to that organ. Vasomotor reflexes that depend on irritation of the corneal nerves can be allayed by the local application of cocaine. External pressure necessary to introduce the needle requires a waiting period until the ocular pressure

has resumed a normal level before satisfactory data may be obtained. When the eye is enucleated, the intra-ocular blood cells are allowed to run out and a sudden drop of pressure occurs due to the emptying of these vessels. This reaches a certain level. It is interesting that this experiment also confirms Dr. Friedenwald's notion that there is no absorption of fluid from the posterior chamber or the anterior chamber as soon as equilibrium is established.

THE ORGANIC SPECIFIC PROPERTIES AND ANTIGENIC POWER IN THE HOMOLOGOUS SPECIES OF ALPHA CRYSTALLIN. DR. ALAN C. WOODS, DR. EARL L. BURKY and DR. M. B. WOODHALL, Baltimore.

The results of experiments were shown indicating that alpha crystallin is the true organic specific substance present in the crystalline lens. Pure alpha crystallin, when injected into animals of the same species, produces antibodies to alpha crystallin obtained from all species studied (rabbits, swine, calves). The presence of beta and gamma crystallin in the extract inhibits this antibody production, which accounts for the conflicting reports of previous observers, and for the failure of many to produce antibodies in animals by injection of homologous lens extracts.

REPORT OF AN EXPERIMENTAL STUDY OF THE REPARATIVE PROCESS OF THE LENS CAPSULE AFTER INJURY. DR. C. A. CLAPP, Baltimore.

Young rabbits were subjected to needling, and the eyes removed for section at intervals up to two months. Slides were shown illustrating the stages in repair of the capsule. The gap is covered by spindle cells, and the elastic membrane covers these over. In one specimen, a typical anterior polar cataract was seen with slight changes in the lens fibers.

CONCERNING A MEMBRANE BETWEEN THE VITREOUS AND ANTERIOR CHAMBER SEEN AFTER REMOVAL OF THE LENS AND ITS CAPSULE. DR. ALFRED COWAN, Philadelphia.

A number of patients were examined with the slit-lamp after intracapsular extraction. On the sixth day after operation, or in some cases sooner, a membrane was seen covering the anterior surface of the vitreous. At first this is herniated through the pupil into the anterior chamber, but it rapidly becomes flatter till it is behind the level of the iris. The vitreous is seen to recede behind this, leaving an optically empty space between its surface and the membrane. This space is seen after from two weeks to several months. The membrane may contain a number of vacuoles that may be close enough together so that vision is markedly reduced. In rare cases shreds of vitreous are seen in front of the membrane, as though there were holes in the membrane. It cannot be considered a layer of condensation of the vitreous-front, as it is separated from the vitreous.

THE CONDITION OF THE OCULAR STRUCTURES IMMEDIATELY AFTER REMOVAL OF THE LENS IN CAPSULE, AS DETERMINED BY MICROSCOPIC EXAMINATION. DR. F. H. VERHOEFF, Boston.

Clear lenses were removed in capsule from two freshly enucleated eyes, and sections were studied. One was a normal eye removed for

orbital neoplasm, the other a leukomatous eye with fluid vitreous, but otherwise without pathologic changes. It was found that the zonular fibers always rupture at their connection with the capsule. The retina and ciliary body were not damaged in any way, and the anterior hyaloid was intact. In the eye with fluid vitreous, the anterior and posterior hyaloid membranes were in contact with each other, almost no vitreous being present between them. This condition is probably more common than is generally believed. The absence of any hyaloid membrane on the retina suggests that the so-called fluid vitreous is really fluid present behind the posterior hyaloid membrane. In degenerated eyes it is known that true vitreous sometimes shrinks into a small mass behind the lens. Whether this condition is always present in eyes with fluid vitreous cannot be stated.

It was concluded that intracapsular extraction by the author's method involves no greater damage to the eye than the capsulotomy method. The advantages of the author's method over other intracapsular methods were brought out.

THE ANTERIOR VITREOUS IN HEALTH AND DISEASE. DR. DANIEL KIRBY, New York.

This article will appear in full in a later issue of the ARCHIVES.

DISCUSSION ON PAPERS OF
DR. COWAN, DR. VERHOEFF AND DR. KIRBY

DR. JOHN W. BURKE, Washington, D. C.: Dr. Burke reported a prolapse of vitreous through the capsule opening, and a thin membrane, which he thought was proliferated, covering the prolapsed vitreous.

DR. JONAS S. FRIEDENWALD, Baltimore: There seems to be no doubt whatsoever that the anterior surface of the vitreous is composed of a membrane, and that this membrane is of such character that it can retain red blood cells. With the evidence so far produced, one cannot conclude irrevocably that it is simply a condensation of vitreous. If it were true that a fragment of vitreous exposed to the aqueous would form a condensation layer on its surface, then the least fragment of vitreous in the anterior chamber should also form a surface membrane around itself. This did not occur.

DR. T. B. HOLLOWAY, Philadelphia: Dr. Holloway recited his experience to support his opinion that this is not condensation, but that it is a definite membrane.

DR. M. J. SCHOENBERG, New York: Dr. Schoenberg, after studying by slit-lamp the surface of the vitreous suspended in water, has been unable to convince himself that there is a hyaloid membrane.

DR. F. H. VERHOEFF, Boston: The origin of what are called membranes is as difficult to explain as the hyaloid membrane. The origin of Descemet's and Bowman's membranes is very obscure. Nobody has given an adequate explanation. Nobody knows how connective tissue is made, or how elastic tissue is made. As a matter of fact, not much is known about anything, and of the vitreous perhaps a little less is known. Some new methods of investigation are needed that will answer these questions. If the vitreous were a gel, it would be the same throughout, and there would be no indication of a hyaloid canal. You

would not find a continuity with the ciliary epithelium. Yet microscopic sections show somewhat similar structure, and that makes me think it is tissue as any other, only differing in degree.

Dr. C. S. O'Brien, Iowa City: Dr. O'Brien subscribed to the idea, conveyed in the three papers that have been read, that there probably is an anterior hyaloid membrane.

Dr. W. H. Wilmer, Baltimore: Dr. Wilmer likes to speak of the hyaloid membrane. Whether it is hyaloid membrane or whether it is a condensation of the outer surface, there is some limiting body that prevents it mixing with the aqueous. Some of the older men call this limiting surface a membrane, and some of the younger men call it a condensation.

Dr. P. C. Jameson, Brooklyn: Dr. Jameson asked how much these membranes contribute to the diminution of vision after cataract operation, whether they should be needled and whether needling favors detachment of the retina.

Dr. Alfred Cowan, Philadelphia: Dogs' eyes have been taken, the cornea and the iris dissected off, and the lens removed. The lens adheres to the patellar fossa probably in the same way as a piece of wet paper would adhere—it may be pulled off. Then this eye is hung in the headrest of the slit-lamp and examined carefully. One can see what undoubtedly is a membrane. You often get a very fine shagreen. Vision is seldom improved by discission of this membrane after intracapsular extraction.

Dr. F. H. Verhoeff, Boston: Dr. Verhoeff has seen only two cases in which discission improved the vision in this type of case.

Dr. Daniel W. Kirby, New York: Dr. Kirby feels that the observers are all in agreement; it is simply a matter of interpretation of the different phenomena that are observed.

Ultraviolet Ocular Therapy. Dr. Lawrence T. Post, St. Louis.

Experiences during five years with various modes of applying phototherapy were reviewed. It was soon found that the Birch-Hirschfeld apparatus with a uviol filter and quartz cell for copper sulphate solution delivered no appreciable amounts of effective ultraviolet rays, almost none below 3,400 angstrom units, which produced no demonstrable changes in the cornea. By removing the uviol filter and substituting distilled water for the copper sulphate solution, light down to 1,800 angstrom units was transmitted. With this light, the dosage must be standardized by testing it on the patient's skin. Exposure for one-half minute usually gives a fair skin reaction, and for one minute, a fairly severe one. Dr. Post usually begins with thirty seconds' exposure, increasing to one minute for later treatments. Although the mercury vapor arc light delivers even more light of short wavelength, the spectrum from the carbon arc as used by Dr. Post seems equally satisfactory. No results were obtained in interstitial keratitis, nodular keratitis, herpes corneae with uveitis, tuberculous uveitis or iritis. In a limited number of conditions, however, definite benefits of irradiation were seen. These were corneal ulcers of the dystrophic type, phlyctenules, interstitial infil-

trates of the cornea and epithelial dystrophy. Vernal conjunctivitis was improved, but not cured.

DISCUSSION

DR. F. H. VERHOEFF, Boston: It was found to be impossible to kill bacteria in the cornea by any ultraviolet irradiation that the human eye could possibly stand. The rays that kill bacteria are stopped on the surface of the cornea, and if there is an ulcer, they are stopped more effectively. So, if one admits that the clinical results have amounted to anything, one must explain the results in a different way than by assuming that there has been direct action on the bacteria.

DR. CLIFFORD B. WALKER, Los Angeles: With two or three treatments with radiation from the mercury vapor lamp, blepharitis is cleared up.

DR. SANFORD R. GIFFORD, Chicago: Dr. Gifford thinks there are only a few conditions subject to beneficial results from the ultraviolet rays, one of which is dendritic ulcer. In lid conditions, such as blepharitis, Kromayer light has been used. In chronic conjunctivitis, if one gives enough roentgen treatments to cause a slight membranous formation, and waits for this to slough off, with two or three such treatments chronic conjunctivitis has been very much benefited.

DR. JOHN GREEN, JR., St. Louis: Dr. Green said that his experience with ultraviolet therapy in ophthalmology has been much more limited than Dr. Post's or Dr. Gifford's, but it has been decidedly favorable. The conditions that have yielded well to this treatment have been marginal ring ulcer and herpes zoster of the cornea.

FURTHER NOTES ON THE METABOLISM OF THE RETINA. DR. FRANCIS H. ADLER, Philadelphia.

This article appears in full in this issue of the ARCHIVES, p. 901.

DISCUSSION

DR. A. MAGITOT, Paris, France: Dr. Magitot discovered that there was no sugar to be found in the anterior chamber of a patient who had had a contusion of the eyeball. After that first finding, he tried some experiments on animals, only a few, and found that in animals that have a larger anterior chamber than man, the quantity of sugar in the aqueous would be considerably lessened after trauma.

DR. JONAS S. FRIEDENWALD, Baltimore: Some years ago, Warburg, measuring the glycolytic activity of various tissues, reported that the glycolysis produced by the retina per cubic millimeter was greater than any other tissue he examined. Adler has rediscovered these facts through a totally different technic, and has confirmed them by several different approaches, thus putting us in the position of being able to reinterpret Warburg's experiments and reestablish their validity.

DR. PETER C. KRONFELD, Chicago: Dr. Kronfeld stated that if a low dextrose content of that part of the vitreous in contact with the retina is due to the high glycolytic power of the retina, then one would expect the vitreous to show all the other characteristics of high glycolysis, namely, high lactic acid and low bicarbonate content. His figures on

lactic acid in the vitreous are not accurate enough to be presented here. The figures presented at the meeting in Washington showed an invariable difference of from 8 to 10 per cent in the bicarbonate content. The bicarbonate content of the vitreous was always lower than the aqueous.

Dr. Kronfeld examined the subretinal fluid in eight cases of retinal detachment, and compared the dextrose content of the aqueous with that of the subretinal fluid. In six of these cases, the dextrose content was considerably lower than that of the aqueous. In two cases the subretinal fluid had almost the same dextrose content as the aqueous, but in those two cases the retina had been pronounced dead.

A retina that has been detached long enough to lose its function entirely apparently has no glycolytic power and therefore does not alter the dextrose content of the fluid around it.

Dr. Francis H. Adler: In closing the discussion, Dr. Adler said that it is of considerable physiologic interest to find out why the retina and brain tissue have such a high rate of glycolysis. It may be that muscle draws on stored glycogen as its fuel, whereas the retina and brain have little dextrose storage and consequently must draw sugar from the circulating blood for their physiologic needs. Unfortunately, his method of sectioning the optic nerve in cats involved the destruction also of blood vessels. Experiments are under way now in which the optic nerve is cut back of the entrance of the central retinal artery, in order that the nutrition of the retina will not be disturbed.

Epipapillary Tissues. Dr. Bernard Samuels, New York.

This article appeared in full in the Archives, October 1931, p. 704.

DISCUSSION

Dr. Parker Heath, Detroit: Some of these tissues are glial tissues rather than proliferates, and may assume a sort of phagocytic property. If there is an imbalance between lipoid and glia tissues, the lipoid may become foreign body in effect. These foreign body lipoids are picked up by the mesenchymal cells.

Dr. Bernard Samuels, New York: In regard to Dr. Heath's comments, Dr. Samuels said that these sections do not permit of the study of lipoids, but it is interesting how much pigment they do contain. Then there are blood pigments. The entire cell must have been dark in life because of the blood pigments.

Measurements of Arteries in Incipient Sclerosis. Dr. F. H. Haessler, Milwaukee.

This article will appear in full in a later issue of the Archives.

DISCUSSION

Dr. Jonas S. Friedenwald, Baltimore: When the eye is examined by the ophthalmoscope and photographed, it is exposed to a considerable amount of light, and the retina is in a state of maximum functional activity. One would therefore expect under these circumstances that the retinal vascular system is in a state of maximum physiologic dilatation, just as one would expect the blood vessels of a muscle contracted at a

maximum rate would be in a state of maximum dilatation. If one could get photographs of the blood vessels of the retina in the dark, one might see considerable change in the influence of drugs, but when the retinal vessels are in a state of maximum physiologic dilatation it seems unlikely that the effect of drugs would make any significant changes.

DR. FRANCIS H. ADLER, Philadelphia: Dr. Adler thinks that there is another reason why Dr. Haessler was unable to show the difference after the administration of amyl nitrite, namely, that while the vessel is not affected particularly by the amyl nitrite, the drug acts on the capillaries and precapillaries which are not photographed; therefore one would not expect to demonstrate all the changes in these photographed vessels. The changes would probably be a reaction on the part of the general circulation or such peripheral changes as might occur in the capillary and precapillary circulation.

DR. A. MAGITOT, Paris, France: It has been pointed out by a few ophthalmologists using tonoscopy that in sclerosis of the retinal vessels, even when the systemic blood pressure is high, there is at the disk a lessening of the arterial blood pressure. It has also been demonstrated that when there is a very high degree of sclerosis of the retinal artery there is generally a very low arterial blood pressure.

THE PRESENCE OF VITAMIN A IN THE RETINA AND CHOROID. DR. A. M. YUDKIN, New Haven.

This article appeared in the ARCHIVES, October, 1931, p. 510.

SOME CASES OF SEPARATION OF THE RETINA AND ITS SUBSEQUENT BEHAVIOR IN NONMYOPIC EYES. DR. F. T. TOOKE, Montreal.

Dr. Tooke believes that in some cases the Gonin method is unnecessarily intricate, and reports his results in four cases in which a less radical procedure was carried out. In one case of traumatic detachment, a reattachment occurred on subconjunctival injections of saline and potassium iodide. Ten years later vision was 5/5 and the field was good.

In the other cases, a 2 mm. trephine opening was made in the sclera, and the cautery applied only to the edges of the scleral incision, which, in two cases, was slightly enlarged with scissors. One patient recovered normal vision, and a second patient a vision of 6/60. In a third case vision of 6/7 was recovered, but the eye was later lost from glaucoma. In three of these cases a tear was seen, which became converted into a white scar. Tooke believes the retina in these cases fell back into place, and was held there as a result of the cauterization.

DISCUSSION

DR. HARRY S. GRADLE, Chicago: Dr. Gradle, after two years' experience with ignipuncture in detachment of the retina, has had results which are between 15 and 20 per cent of reattachment following the operation. How much of this is due to the operation and how much to the beneficial forces of nature he is not prepared to say. He is obtaining results following ignipuncture, where no tear is visible, and where the cautery is used at the apex of the elevation. He wished to record a case he has just had of detachment, where a perfectly cleancut tear became visible

after the detachment had appeared. The patient objected to operation, consequently was put to bed, and the detachment disappeared spontaneously in the course of three or four days. The patient had a high blood pressure.

DR. A. MAGITOT, Paris, France: Dr. Magitot feels that all will agree that these tears in the retina are not found so often as Gonin thinks they are. His experience showed him that one cannot find these tears in the retina in more than from 50 to 60 per cent of cases, even when examining the patients carefully.

He does not share entirely Gonin's view, his detachment theory, because what Gonin calls subretinal fluid is not in fact subretinal fluid, it is intraretinal, and if one examines the retina, one is bound to say that this detachment of the retina is in reality a cyst of the retina. And there is another thing. The fluid inside these cysts is quite different from that of the vitreous. Dr. Magitot examined many of these fluids, and it contains such a high degree of albumin that it is impossible that it is vitreous. One can puncture the vitreous ten times and never find such a high degree of albumin as was found in this subretinal or intraretinal fluid.

DR. EDWARD C. ELLETT, Memphis: The idea has been expressed that the small detachments, the flat detachments, are amenable to treatment with the cautery, whereas the larger detachments are more suitable for subconjunctival injection, which is actually the idea of which Dr. Tooke speaks.

DR. JONAS S. FRIEDENWALD, Baltimore: Dr. Friedenwald applied the Gonin operation in nine cases. In two there has not been reattachment, but vision is enormously improved. Three were failures. No two cases of detachment are alike, and no one has as yet been able to approach any sort of classification by which the results of any operation can be shown.

DR. WILLIAM C. FINNOFF, Denver: Dr. Finnoff believes that retinal cysts frequently precede holes in the retina. In the eight cases he has studied carefully, cysts were found in the retina in four. If multiple cysts are found, he thinks that the prognosis is apt to be poor. It might be worth while, where large cysts are found, also to cauterize this area to prevent the formation of holes at a later period.

DR. SANFORD R. GIFFORD, Chicago: Dr. Gifford was interested in Dr. Tooke's results and opinions which agreed with those arrived at by Meller, Guist and others in an explanation of the effect of the cauterization. This cannot be due to actual searing of the retina except in the case of very small holes, but must be due to the formation of a plastic exudate on the choroid, to which the retina adheres when the subretinal fluid is removed. Localization of the hole is important, as, if the retina adheres over an area including the hole, the connection between the vitreous and subretinal space is sealed off and the attachment is more apt to persist. The technic of chemical cauterization to bring about this result, as practiced by Guist, was described.

DR. PETER C. KRONFELD, Chicago: Dr. Kronfeld stated that if we confine our report of tears to certain sizes, we get very different results. Here each of us can report from ten to twenty-five cases, and among these are retinal detachments of every origin, every etiology, and from a

different standpoint of treatment, so our results cannot be compared at all. Dr. Kronfeld showed a Guist pencil, a thin stick of potassium hydroxide covered by a waxlike material in order to prevent contact with the humidity of the atmosphere.

DR. F. H. VERHOEFF, Boston: Dr. Verhoeff felt that it is difficult to be sure whether the hole comes after the separation or is the cause of the separation. It is known that in senile eyes this cystoid condition is very common, so he thinks it is probable that in a majority of cases in which one finds a hole, it is the cause of the separation rather than the result. One can get tears in the retina from direct trauma, and it is probable that one can get a tear of the retina simply from constant movement of the eye where there is a large separation.

Dr. Verhoeff felt that Gonin's idea is perfectly logical, and that the results of the operation seem to bear out his views.

ANALYSIS OF THE PROLIFERATIVE AND EXUDATIVE ELEMENTS IN SOME CASES OF SYMPATHETIC OPHTHALMIA. DR. HARVEY LAMB, St. Louis.

An histologic study of the proliferative and exudative processes on the free surfaces of the uveal tract in six cases of sympathetic ophthalmia was given.

Brief mention was made of the findings and opinion of Schirmer, Ruge and Fuchs, but particularly of Ruge, regarding the significance of the fibrinoplastic exudate in sympathetic ophthalmia.

This was followed by a report of the proliferative changes in one case of the specific sympathetic cells from the iris and choroid into the open wounds made by a transfixing gunshot, in another case of the same changes beyond the choroid and in contact with a necrotic melanosarcoma, and similar changes in a third case on the free surfaces of the iris and ciliary body associated with iritic and cyclitic membranes. In the fourth case proliferative processes along the posterior emissary vessels, and later inward within the sclera, probably accounted for the production of true sympathetic disease after a panophthalmitis and evisceration. The fifth case with no involvement of the fellow eye was found indicative of sympathetic uveitis according to Fuchs; with a vitreous abscess there was present considerable proliferation of the ciliary epithelium and the retinal pigment epithelium. The last case of sympathetic ophthalmia followed an iridectomy for glaucoma due to a large melanosarcoma of the choroid; a hypopyon composed entirely of eosinophil leukocytes was found.

In these few cases the principal changes on the surface of the uveal tract, as well as within it, are proliferative ones.

DISCUSSION

DR. E. V. L. BROWN, Chicago: Dr. Brown stated that five cases could not be considered as offering any evidence as to the pathology of sympathetic ophthalmia, since they showed the changes of ordinary fibrinoplastic uveitis. The sixth case was more interesting, but Brown does not believe it presents the picture described by Fuchs. The plasma cells are not characteristic of sympathetic ophthalmia, and the vitreous abscess is evidence against it. No "cell nests" were seen. He believes

that the paper offers no evidence that casts doubt on the characteristic picture of sympathetic ophthalmia.

DR. F. H. VERHOEFF, Boston: Dr. Verhoeff believes that there is a characteristic picture of sympathetic ophthalmia. From examination of the section he does not think that the sixth case was one of sympathetic ophthalmia. He mentioned having made the diagnosis of sympathetic ophthalmia from sections when the patients knew of no trouble in the second eye, and that on sending for these patients he found them to show a quiet uveitis beginning.

CONCERNING THE ANALOGIES AND DIFFERENCES BETWEEN CHOKED LABYRINTH, GLAUCOMA AND CHOKED DISK. DR. CLIFFORD B. WALKER, Los Angeles.

The anatomy of the eye and internal ear was reviewed to point out analogies in the circulation of fluid in both organs. The processes concerned in choked labyrinth were compared to those in glaucoma.

BLINDNESS AND CHOKED DISK IN GUERNSEY BULL CALVES. DR. GEORGE E. DE SCHWEINITZ, Philadelphia.

This article will appear in full in a later issue of the ARCHIVES.

DISCUSSION

DR. WILLIAM TARUN, Balitmore: Dr. Tarun had seen four similar cases in a Guernsey herd. All showed choked disk, one in only one eye, and one had retinal hemorrhage.

OPTIC NEURITIS DUE TO DISEASE OF THE SPHENOIDAL SINUSES. DR. ALBERT E. BULSON, Fort Wayne, Ind.

The literature on this subject was reviewed, and personal cases showing a definite relationship between the two conditions were cited. Dr. Bulson took exception to Cushing's statements, and believes papilledema may be caused by sphenoiditis.

DISCUSSION

DR. T. B. HOLLOWAY, Philadelphia: Dr. Holloway reported two cases with choked disk and sinusitis in which no intracranial pathologic process was ever found. He believes that it is illogical to assume multiple sclerosis as a cause of neuritis unless other symptoms are present.

DR. F. H. VERHOEFF, Boston: Dr. Verhoeff believes that a similar condition is present in all typical cases and that the cause is probably multiple sclerosis, which may involve the nerve alone. Papilledema may be due to inflammation of the nerve near the globe, with multiple sclerosis as the cause.

DR. E. V. L. BROWN, Chicago: Dr. Brown reported a case with a definite relationship to sphenoiditis, but thinks such cases must be rare.

DR. JOHN GREEN, St. Louis: Dr. Green saw a case in which choked disk improved following operation for sphenoiditis, but a cyst of the brain was later found.

DR. CLIFFORD B. WALKER, Los Angeles: Dr. Walker tried salicylates in acute retrobulbar neuritis, and gave as much as 75 grains (4.87

Gm.) a day. Improvement stopped when the salicylates were stopped. In two or three weeks he used the salicylates again, and improvement began again. He felt that there is some similarity in the various infections that do respond well to the salicylates.

DR. A. D. PRANGEN, Rochester, Minn.: It has not been Dr. Prangen's experience to encounter sinus disease associated with disease of the optic nerve. His cases have been due apparently to early multiple sclerosis, with other toxic conditions such as the alkaline disturbances of pregnancy. He feels that the etiology of acute retrobulbar neuritis deserves more study.

ENCEPHALITIS AFTER MEASLES. DR. PARKER HEATH, Detroit.

Among fifty-five cases of encephalitis in a Detroit hospital, Dr. Heath found eleven due to measles. The only case showing fundus changes was in a boy of 6 with poor vision developing on the eleventh day of the disease. The child was apparently blind when first seen, with wide immobile pupils, pallor of the disks and threadlike arteries. Exudate developed in the macula and a fine displacement of pigment in the periphery of both fundi. Under injections of typhoid vaccine and hot packs with the electric blanket, a gradual dilatation of the vessels was observed. Vision returned to normal after several weeks, and only fine macular changes remained.

DISCUSSION

DR. T. B. HOLLOWAY, Philadelphia: Dr. Holloway saw one case of encephalitis, due to measles, with bilateral optic neuritis.

ALBINISM IN THE PROGENY OF NEGRO AND WHITE PARENTS. DR. CHARLES M. SWAB, Omaha.

Dr. Swab's patient was the son of a white father who was married to a Negress. A black daughter was born, and the albino child was the result of incestuous union between father and daughter. The child had negroid features and hair, but was a complete albino. Nystagmus was present, the disks were pale and vision was reduced to fixing a light at 1 meter. The fundi were myopic 8 diopters and showed no trace of pigment. Theories of albinism were reviewed, none apparently agreeing with all the facts.

As an aid to vision in such cases, the use of opaque contact glasses with a small hole for the pupil was suggested.

DISCUSSION

DR. WILLIAM ZENTMAYER, Philadelphia: Dr. Zentmayer saw three albino children in one Negro family.

SUBNORMAL ACCOMMODATION. DR. A. D. PRANGEN, Rochester, Minn.

This article appears in full in this issue of the ARCHIVES, p. 906.

DISCUSSION

DR. C. D. WESCOTT, Chicago: Dr. Wescott reported that some patients recover quickly from the effects of homatropine, some more

slowly. Dr. Duane always insisted that if one allowed a week between the examination following the use of homatropine and the post cycloplegic test, it would save difficulty. In all cases of subnormal accommodation, he has insisted on general physical examination and the greatest possible care of the general health. He also has prescribed bifocal glasses for patients in the early twenties who were obliged to use their eyes closely all day.

DR. W. CAMPBELL POSEY, Philadelphia: Dr. Posey stated that Dr. Dyer's method of treating subnormal accommodation consisted of lengthening periods of reading, beginning with a few minutes and gradually accustoming the patient to longer periods of reading, and in that way building up his accommodative power. In his "History of Ophthalmology," Dr. Hirschberg called attention to the fact that Dr. Dyer had been the first to point this out, and that Dr. Graefe himself had referred to it.

DR. EVERETT L. GOAR, Houston, Texas: Dr. Goar asked Dr. Prangen to explain his classification of dynamic and static subnormal accommodation.

DR. A. D. PRANGEN, Rochester, Minn.: Dr. Prangen stated that it is to be regretted that accurate professional workers in this field cannot be obtained. He wished to acknowledge the achievements of Jackson and Duane in their monumental piece of work in establishing the accommodative normal values for the different age periods, without which he would be unable to practice.

The classification asked for is that of Dr. Duane, and is to be found in his article in the ARCHIVES OF OPHTHALMOLOGY (**54:** 233 [May] 1925).

Book Reviews

Augenärztliche Eingriffe. By Prof. J. Meller. Third edition. Price, 28 marks. Pp. 352, with 199 illustrations. Berlin: Julius Springer, 1931.

The new (third) edition of Meller's excellent "Operative Surgery" maintains the same high standard that was conspicuous in previous editions. It considers only the methods that are in actual use in the First Eye Clinic of the Vienna General Hospital, methods constructed on principles that have stood the test of time; thus, the beginner starts with a simplified and firm foundation. In no other book will the reader find so much information on so many important details; all this arises from the large experience Professor Meller has gained in many years of teaching.

Two new chapters have been inserted, one on cataract extraction in-the-capsule and another on ignipuncture for retinal detachment.

The description of the operation of cataract extraction in-the-capsule is not the best. After grasping the cataract at its lower surface with a Stanculeanu or an Elschnig forceps (no mention is made of the Kalt forceps, which is better than either of the other two), the cataract is dislocated and drawn out, the forceps remaining in situ. The disadvantages of this method have been definitely demonstrated, and the procedure of "tumbling" the lens, which method offers the greatest degree of safety, is entirely overlooked. The advantages of an iridectomy in this operation are not sufficiently appreciated, and the reviewer is not convinced of the necessity of an anesthetic retrobulbar injection, with its consequent softening of the eyeball.

The chapter on the operative treatment for detachment is sane and excellent. The description of the difficulties, complications and general deductions is most timely and instructive. The tears and holes in the retina in detachment are first described; then follow the methods for their localization, with resulting errors, and the methods of operating. The author uses a point galvanocautery at white heat, perforating the sclera, introducing the tip only to a depth of 2 mm. and for not longer than five seconds. The complications are early and late hemorrhages, an inflammatory reaction, glaucoma, scar contraction and the development of new tears. Gonin's method with the Paquelin thermocautery and Larson's endothermia are also described. Meller regards the latter method with favor, and believes that it is of value particularly in combination with ignipuncture, especially in detachments in which no hole can be found. He concludes that ignipuncture should be tried only in recent cases; more than one perforation is not advisable at a sitting. Contraindications are opacities of the media, holes that are too large or inaccessible, reduced ocular tension and inflammation of the anterior eye segment.

The present English translation of Meller's "Operative Surgery" has been so useful and deservedly popular with English readers that an early translation of this new edition should be promptly forthcoming.

A. K.

Results of Etiologic-Morphologic Investigations in Trachoma and Allied Inclusion Diseases. By J. R. Thim. Pp. 43, with 4 colored plates. Pécs, Hungary: Dunantul Buchdruckerie, 1930.

The work reported in this monograph was carried out in 1919 and later in the Government Culture-Collection, the First Eye-Clinic in Vienna and the University Clinic at Pécs, Hungary.

In 1919, the author studied preparations from three cases of inclusion urethritis, and found what he considered to be vegetative forms of a protozoan, with free cilia. He was able to follow stages in its development, including an encysted stage. Similar findings were made in the female genitalia, in inclusion blennorrhea of the new-born and in inclusion conjunctivitis of adults. In the last two diseases, bodies were found that resembled developmental stages of Trichomonas. Protozoan bodies were also found in a case of swimming-pool conjunctivitis. Material from untreated trachoma was then investigated, and similar pictures were seen.

In preparations from the urethral and vaginal mucosa, bodies were found both free and in the cells, with the protoplasm staining blue with the Giemsa stain, and containing many red-staining granules, with a vacuolated nucleus and two large cilia. What were considered encysted forms of this supposed protozoan were also found, with radially arranged spores. These became free, and in this stage were called elementary bodies. These bodies were supposed to enter the epithelial cells again and to form inclusions like those seen in trachoma. These must be distinguished, as in trachoma, from intracellular débris of various kinds, called pseudoinclusions.

The same bodies, considered by Thim as protozoans, were found in the conjunctional secretions of the new-born, and in cases of inclusion blennorrhea. In swimming-pool conjunctivitis and in one case of urethritis, he found bodies that he thinks are different from those previously described, and different from each other. Apparently the bodies seen in trachoma are also different from those of inclusion blennorrhea and urethritis, and Thim considers them due to two distant protozoans. In trachoma he was never able to demonstrate encysted bodies, which he considers an essential difference from the findings in inclusion blennorrhea.

The author's conclusions rest on his findings in three cases of urethritis and vaginitis, three of inclusion conjunctivitis, one of swimming-pool conjunctivitis and ten of trachoma.

A number of good colored cuts and three photomicrographs accompany the monograph. Evidently, cilia were seen only in preparations from the genitalia, and these forms and certain others suggested to the author a possible relation of the parasite to Trichomonas.

S. R. GIFFORD.

Report of the Chengtu Eye, Ear, Nose and Throat Hospital, Chengtu, West China, 1929-1930.

This mission hospital was organized in 1928, and work was begun in 1929, in buildings previously used for a women and children's clinic. The hospital is the teaching and clinical unit for ophthalmology

and otolaryngology of the West China Union University. Dr. R. A. Peterson, who is director of the hospital, is head of both departments in the university and has associated with him Dr. Lo and Dr. Lu. Dr. E. R. Cunningham has recently been sent out from Moorfields Hospital to develop especially the work in ophthalmology.

The outpatient department receives more than 50,000 visits, and the hospital houses 1,000 patients annually. The ophthalmologic department is well equipped with modern instruments for diagnosis and treatment, including the Gullstrand ophthalmoscope, the slit-lamp and the irradiation lamp.

In spite of some radical agitation, culminating in a severe knife wound inflicted by a patient on Dr. Peterson, the community as a whole has recognized the value of the work done, as shown by an increase in clinic attendance from 39,500 visits in 1929 to 56,861 in 1930. Nearly 1,500 major and minor operations were performed during 1930.

Trachoma is present in 25 per cent of the population, and in from 50 to 60 per cent of the patients. Expression, with the application of phenol or trichloracetic acid followed by persistent use of copper sulphate, is the usual treatment followed. In cases of ectropion, linear incision of the tarsus combined with marginoplasty by implantation of mucous membrane from the lip has proved satisfactory. Dacryocystitis is frequent and is treated by probing and destruction of the sac with trichloracetic acid, and the Mosher-Toti procedure in selected cases. Keratitis from smallpox is frequent and very severe. Forty-seven patients with cataracts were operated on in 1930, a small number compared with those seen in the Indian clinics.

Lectures are given to the fourth year medical students; part of the rotating internship is spent in the hospital during the fifth year, and a number of men have spent varying lengths of time in postgraduate work. By taking private and semiprivate native patients, as well as a large number of free patients, the work has been made largely self-supporting.

The importance of this type of modern service in the specialties in an area so isolated that some patients must travel three weeks to reach the hospital can be easily understood.

S. R. GIFFORD.

Annual Report of the Government Ophthalmic Hospital, Madras, India, for the Year 1930. By Lt. Col. R. E. Wright, M.D., Superintendent. Price, 12 annas.. Pp. 26. Madras: Government Press, 1931.

During the past year, 3,720 inpatients and 28,470 outpatients were treated. Among more than 3,000 operations, there were 1,676 cataract extractions, of which 1,327 were done with a capsulotomy. In these there were 18 cases of vitreous escape and 15 cases of iris prolapse. The Barraquer procedure was employed in 218 extractions, with vitreous escape in 8 and iris prolapse in 4. The bridge flap was adopted in the majority of cases; it is followed by a denser after-cataract, and the

reformation of the anterior chamber is delayed, which Wright associates with adhesions of capsular tags to the corneal endothelium and consequent keratitis.

Trephining for glaucoma was done in 117 cases; Lagrange, or its modification, was adopted in 30 cases. There were several cases of trephining in which the central vision was very good, but the fields were reduced to within a few degrees of the fixation point. These cases retained their fields and visions after operation.

Superficial punctate keratitis was seen in even a greater number of cases than in previous years, and the disease has taken on a somewhat different aspect, with more conjunctivitis. Deep changes of the cornea of a milder degree were also observed more frequently, such as fine corneal precipitates. There was no iritis. The severity of the signs still remained out of proportion to the symptoms. A search for possible organisms has been without result.

The patients with keratomalacia were treated with vitamin A, though it did not seem as effective as cod liver oil. Chaulmoogra oil was used in the treatment for trachoma, and though it gave considerable relief and was not disagreeable, the results seemed to be identical with those of the older forms of treatment. Twenty-nine patients were treated with radium.

Wright and his co-workers are to be congratulated on the continuation of the splendid work they are doing in India.

A. K.

The Movements of the Eyes in Reading. By M. D. Vernon. Report of the Committee on the Physiology of Vision, Medical Research Council. Price, 9 pence. Pp. 45. London: His Majesty's Stationery Office, 1930.

In continuing her work on the "Psychology of Reading" (number 130 of the same series), the author investigated the effects of various psychologic factors on the effector mechanism of reading. The effects were judged by studying the ocular movements, and were recorded by an apparatus that photographed a beam of light reflected from the cornea on a moving film.

It was established that the eye did not remain steady during voluntary fixation. Voluntary movements were not performed accurately, as the eye usually stopped short of the correct point, but occasionally overshot it. Kinesthetic sensations did not give an accurate impression of eye movement.

Disconnected words were read at a slow and variable rate. Meaningful material, however, was read at a rate that varied with the attitude of the subject to that material. Reading was rapid and regular when the subject was uninterested in the meaning of the passage, and rapid and somewhat irregular when the subject was moderately interested or when the trains of associated ideas or imagery were of no personal interest. The rate was slow when the subject was interested, particularly if difficulty in comprehending the meaning was encountered. It was slow and irregular when a train of thought associated with strong personal interest was excited.

This report embodies the result of ingenious and painstaking research and is an interesting addition to the series sponsored by the Committee on the Physiology of Vision.

G. M. BRUCE.

Hereditary Optic Atrophy (Leber's Disease). By Julia Bell. The fourth memoir, Anomalies and Diseases of the Eye, being the second volume of The Treasury of Human Inheritance, issued by Francis Galton Laboratory of Eugenics, under the editorship of Prof. Karl Pearson. Price, 36 shillings. Pp. 325-423. London: Cambridge University Press, 1931.

In Leber's disease there is a larger number of fully worked out pedigrees than in any other condition. Though this disease has long been known, it was not fully worked up until Leber's publication appeared in 1871. The author is inclined to doubt the accuracy of the statement that its transmission is sex-limited. There is a marked responsibility of females in regard to the transmission of the disease, and if the sisters of the affected males were to abstain from parentage, the disease would be almost exterminated. The age of onset in males is between 20 and 23; in females, between 12 and 15, and 24 and 27. The diminution of vision is the only symptom, and, in rare cases, blindness. The central scotoma interferes with direct vision and fine work of the eyes. The prognosis is generally unfavorable. Complete recovery is infrequent, though improvement has been observed in some cases. Rehsteiner (*Arch. f. Ophth.*) is the only one who has given an anatomic examination of a case in question. This author found atrophy of the ganglion cells and nerve fibers of the retina in the macular region and in a large part of the surrounding retina, including the papillomacular bundle, together with a less uniform atrophy of the fibers of the optic nerve. There was no evidence pointing to an inflammatory origin.

(From Brit. M. J. 1:1119 [June 27] 1931.)

Directory of Ophthalmologic Societies *

FOREIGN

British Medical Association, Section on Ophthalmology

President: Mr. E. E. Maddox, Glenartney, Poole Road, Bournemouth, England.

Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford. Time: July 7-9, 1932.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.
Place: Paris. Time: July 17-21, 1932.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.
Place: Leipzig. Time: June, 1932.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Rotterdam, Holland.
Place: Madrid, Spain. Time: 1933.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Parker Heath, 1551 Woodward Ave., Detroit.
Place: New Orleans. Time: May 11-13, 1932.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal, Canada.
President-Elect: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Montreal, Canada. Time: 1932.

American Ophthalmological Society

President: Dr. Edward C. Ellett, 130 Madison Ave., Memphis, Tenn.
Secretary: Dr. Emory Hill, Professional Bldg., Richmond, Va.
Place: New London, Conn. Time: June, 1932.

SECTIONAL

New England Ophthalmological Society

President: Dr. S. Judd Beach, 704 Congress St., Portland, Me.
Secretary-Treasurer: Dr. E. B. Dunphy, 520 Commonwealth Ave., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. W. F. Hoffman, 317 Summit Ave., Seattle.
Secretary-Treasurer: Dr. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: Seattle. Time: June 30-July 2, 1932.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Albert E. Hillis, 1115 Pacific Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Sioux Valley Eye and Ear Academy

President: Dr. John B. Gregg, 101 S. Main St., Sioux Falls, S. Dakota.
Secretary-Treasurer: Dr. F. H. Roost, 420 Trimble Bldg., Sioux City, Iowa.
Place: Sioux City, Iowa. Time: November, 1932.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Wiley R. Buffington, 211 Camp St., New Orleans.
Secretary: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Birmingham, Ala. Time: November, 1932.

Southwestern Michigan Triological Society

President: Dr. C. Ward Ellis, 215 N. Walnut St., Lansing.
Secretary: Dr. H. B. Weinburgh, 301 Seymour St., Lansing.
Time: Third Thursday of alternate months.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Ralph W. Danielson, 258 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the Month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. M. H. Merriman, 115 Prospect St., Waterbury.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. W. L. McDougall, 50 Whitehall St., S. W., Altanta.
Secretary-Treasurer: Dr. W. O. Martin, Jr., 412 Medical Arts Bldg., Atlanta.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. R. Gillum, 221 S. 6th St., Terre Haute.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. Gordon F. Harkness, 215 Main St., Davenport.
Secretary-Treasurer: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Wilfred Haughey, 303 Post Bldg., Battle Creek.
Secretary: Dr. H. O. Westervelt, 239 Pipestone St., Benton Harbor.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Douglas F. Wood, 74 S. 9th St., Minneapolis.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. L. H. Huber, Livingston.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. F. L. Wicks, Valley City.
Secretary-Treasurer: Dr. W. L. Diven, National Bank Bldg., Bismarck.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. M. Hendershott, 193, 11th St., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Frank W. Dimmitt, 195 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. D. Jones, 3116 Live Oak St., Dallas, Texas.
Secretary: Dr. A. F. Clark, 1034 Nix Bldg., San Antonio.
Place: San Antonio. Time: December, 1931.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. D. W. Henderson, 75 S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. T. F. Welch, 14 Exchange Pl., Salt Lake City.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. C. P. Jones, Newport News.
Secretary-Treasurer. Dr. H. G. Preston, 307 Professional Bldg., Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. G. A. Smith, Montgomery.
Secretary: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Charles W. Buvinger, 50 Washington St., East Orange.
Secretary: Dr. Earl LeRoy Wood, 192 Roseville Ave., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ACADEMY OF MEDICINE OF TORONTO, SECTION OF OPHTHALMOLOGY

Chairman: Dr. P. J. F. Houston, 34 Hurndale Ave., Toronto.
Secretary: Dr. Alexander E. MacDonald, 151 Bloor St., W., Toronto.
Time: First Monday of winter months.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John R. Childs, 45 Edgewood Ave., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Alan C. Woods, 906 Cathedral St., Baltimore.
Secretary: Dr. Henry F. Graff, 513 N. Charles St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. John N. Evans, 23 Schermerhorn St., Brooklyn.
Secretary: Dr. Michael J. Buonaguro, 589 Lorimer St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday of February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. A. F. Luhr, 40 W. North Ave., Buffalo.
Secretary-Treasurer: Dr. Thurber Le-Win, 112 Linwood Ave., Buffalo.

Chicago Ophthalmological Society

President: Dr. Frank E. Brawley, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. William V. Mullin, 9204 Euclid Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 2064 E. 9th St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. Roy B. Metz, Hanna Bldg., Cleveland.
Secretary: Dr. Paul G. Moore, Medical Arts Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Maxwell Langdon, 1530 Locust St., Philadephia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Ivor Clark, 188 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Francis W. Thomas, 327 E. State St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. M. Sellers, 717 Pacific Ave., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, 717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. Cecil C. Jones, 1121 Equitable Bldg., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Parker Heath, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. F. M. Sulzman, 1831, 5th Ave., Troy.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1011 Medical Arts Bldg., Fort Worth.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, 26 Sheldon Ave., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 26 Sheldon Ave., Grand Rapids, Mich.
Place: Various Local Hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Claude C. Cody, 1306 Walker Ave., Houston, Texas.
Secretary: Dr. J. M. Robison, Eye, Ear, Nose and Throat Hospital, Houston.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. B. J. Larkin, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. R. R. Montgomery, 102 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Russell T. Uhls, 823 Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section on Ophthalmology and Oto-Laryngology

President: Dr. Eugene R. Lewis, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Lutheran Hospital, Assembly Room. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Herbert F. Wolters, 130 Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otological Society

President: Dr. W. R. Buffington, 211 Camp St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. William P. Haney, 107 S. 17th St., Omaha.
Vice President: Dr. L. G. Howard, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassiday, 1620 Medical Arts Bldg., Omaha.
Place: University Club, 1914 Harney St., Omaha. Time: 6. p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Emanuel U. Wallerstein, Professional Bldg., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. Clyde Heatly, 11 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 S. Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Assn., 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Martin H. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: St. Louis or Washington University Cafeteria.
Time: 6:30 p. m., fourth Friday of each month from October to April, inclusive.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Scott C. Applewhite, 705 E. Houston St., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. W. F. Swett, 490 Post St., San Francisco.
Secretary: Dr. Robert C. Martin, 384 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. Claude L. La Rue, Highland Clinic, Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 505 Medical Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Paul A. Remington, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. M. G. Brown, 713 E. Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy Dunlap, 108 W. 6th St., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, 108 W. 6th St., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.

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